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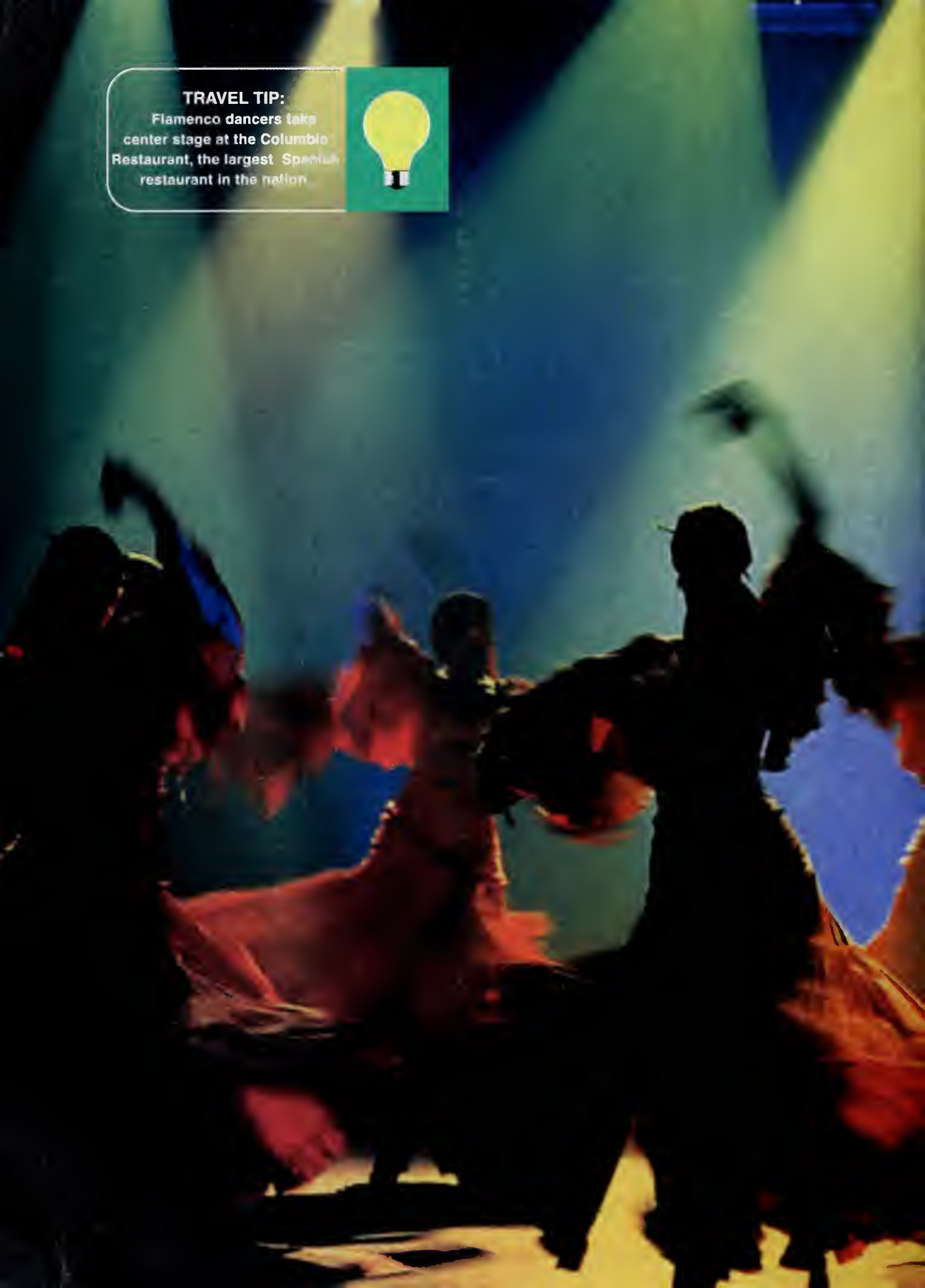
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Congenital Cardiovascular Disease in the Adult Patient: Imaging Update—Steiner RM, Reddy GP, Flicker S. J Thorac Imaging 2002 Jan;17(1):1-17.

Tiotropium in the Management of Chronic Obstructive Pulmonary Disease (editorial)—Rees PJ. Eur Respir J 2002 Feb;19(2):205-206.

Exercise Gas Exchange, Breath-by-Breath—Wasserman K. Am J Respir Crit Care Med 2002 Feb 1;165(3):325-326.

Asthma in the Transition from Childhood to Adulthood—Russell G. Thorax 2002 Feb;57(2):96-97.

The Imaging Spectrum of Pulmonary Tuberculosis in AIDS—Saurborn DP, Fishman JE, Boiselle PM. J Thorac Imaging 2002 Jan;17(1):28-33.

Weaning Protocols: Here to Stay (commentary)—Keenan SP. Lancet 2002 Jan 19;359(9302):186-187.

A 55-Year-Old Patient with Advanced COPD, Tracheostomy Tube, and Sudden Respiratory Distress—Hodder RV. Chest 2002 Jan;121(1):279-280.

Clinical Practice: Treatment of Tobacco Use and Dependence—Rigotti NA. N Engl J Med 2002 Feb 14;346(7):506-512.

Optimizing Sedative Use in the Intensive Care Unit—Ferguson ND, Mehta S. Intensive Care Med 2002 Jan;28(1):44-47.

Asthma in Exercising Children Exposed to Ozone: A Cohort Study—McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, et al. Lancet 2002 Feb 2;359(9304):386-391.

BACKGROUND: Little is known about the effect of exposure to air pollution during exercise or time spent outdoors on the development of asthma. We investigated the relation between newly-diagnosed asthma and team sports in a cohort of children exposed to different concentrations and mixtures of air pollutants. METHODS: 3535 children with no history of asthma were recruited from schools in 12 communities in southern California and were followed up for up to 5 years. 265 children reported a new diagnosis of asthma during follow-up. We assessed risk of asthma in children playing team sports at study entry in six communities with high daytime ozone concentrations, six with lower concentrations, and in communities with high or low concentrations of nitrogen dioxide, particulate matter, and inorganic-acid vapour. FINDINGS: In communities with high ozone concentrations, the relative risk of developing asthma in children playing three or more sports was 3.3 (95% CI 1.9-5.8), compared with children playing no sports. Sports had no effect in areas of low ozone concentration (0.8, 0.4-1.6). Time spent outside was associated with a higher incidence of asthma in areas of high ozone (1.4, 1.0-2.1), but not in areas of low ozone. Exposure to pollutants other than ozone did not alter the effect of team sports. INTERPRETATION: Incidence of new diagnoses of asthma is associated with heavy exercise in communities with high concentrations of ozone, thus, air pollution and outdoor exercise could contribute to the development of asthma in children.

Excessive Erythrocytosis, Chronic Mountain Sickness, and Serum Cobalt Levels—Jefferson JA, Escudero E, Hurtado ME, Pando J, Tapia R, Swenson ER, et al. Lancet 2002 Feb 2;359(9304):407-408.

In a subset of high-altitude dwellers, the appropriate erythrocytotic response becomes excessive and can result in chronic mountain sickness. We studied men with (study group) and without excessive erythrocytosis (packed-cell volume >65%) living in Cerro de Pasco, Peru (altitude 4300 m), and compared them with controls living in Lima, Peru (at sea-level). Toxic serum cobalt concentrations were detected in 11 of 21 (52%) study participants with excessive erythrocytosis, but were undetectable in high altitude or sea-level controls. In the mining community of Cerro de Pasco, cobalt toxicity might be an important contributor to excessive erythrocytosis.

Improved Health Outcomes in Patients with COPD During 1 Yr's Treatment with Tiotropium—Vincken W, van Noord JA, Greefhorst AP, Bantje TA, Kesten S, Korducki L, Cornelissen PJ; Dutch/Belgian Tiotropium Study Group. Eur Respir J 2002 Feb;19(2):209-216.

Tiotropium, a novel once-daily inhaled anticholinergic, has been shown to improve lung function over a 24-h period. In order to extend these findings, health-outcomes were evaluated over 1 yr in chronic obstructive pulmonary disease (COPD) patients. Spirometric results, peak expiratory flow rate (PEFR), salbutamol use and effects on dyspnoea, health-related quality of life and COPD exacerbations were assessed in two identical 1-yr randomized double-blind double-dummy studies of tiotropium 18 µg once daily (n=356) compared with ipratropium 40 µg q.i.d. (n=179).

Screening forced expiratory volume in one second (FEV_1) were 1.25±0.43 L (41.9±12.7% of the predicted value) (tiotropium) and 1.18±0.37 L (39.4±10.7% pred) (ipratropium). Trough FEV_1 at 1 yr improved by 0.12±0.01 L with tiotropium and declined by 0.03±0.02 L with ipratropium ($p<0.001$). Significant improvement in PEFR, salbutamol use, Transition Dyspnea Index focal score, and the St George's Respiratory Questionnaire total and impact scores were seen with tiotropium ($p<0.01$). Tiotropium reduced the number of exacerbations (by 24%, $p<0.01$), and increased time to first exacerbation ($p<0.01$) and time to first hospitalization for a COPD exacerbation ($p<0.05$) compared with ipratropium. Apart from an increased incidence of dry mouth in the tiotropium group, adverse events were similar between treatments. Tiotropium was effective in improving dyspnoea, exacerbations, health-related quality of life and lung function in patients with chronic obstructive pulmonary disease, and exceeds the benefits seen with ipratropium. The data support the use of tiotropium once-daily as first-line maintenance treatment in patients with chronic obstructive pulmonary disease.

A Long-Term Evaluation of Once-Daily Inhaled Tiotropium in Chronic Obstructive Pulmonary Disease—Casaburi R, Mahler DA, Jones PW, Wanner A, San PG, ZuWallack RL, et al. Eur Respir J 2002 Feb;19(2):217-224.

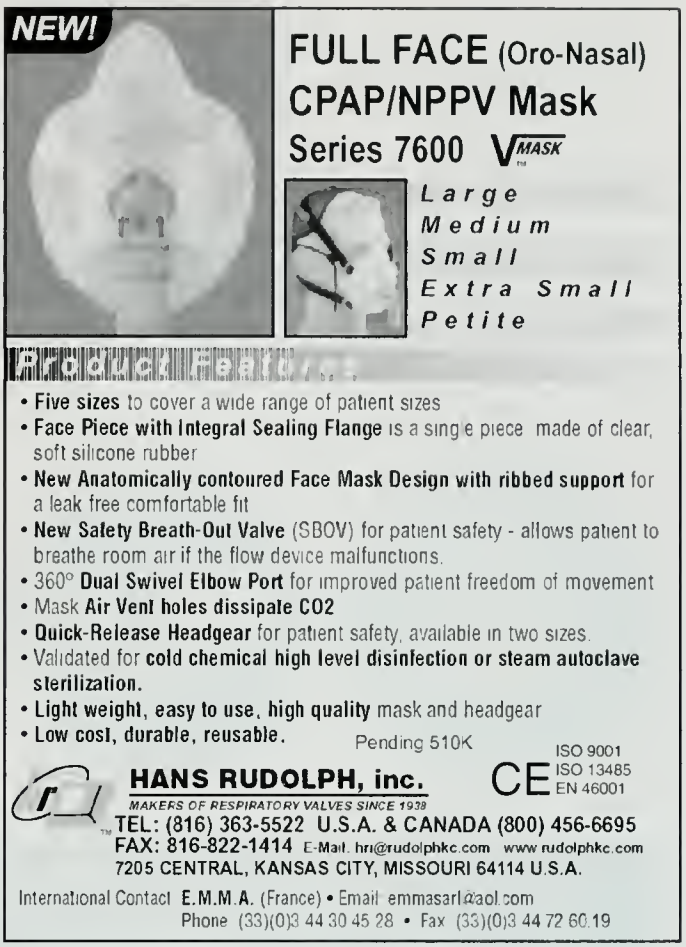
Currently available inhaled bronchodilators used as therapy for chronic obstructive pulmonary disease (COPD) necessitate multiple daily dosing. The present study evaluates the long-term safety and efficacy of tiotropium, a new once-daily anticholinergic in COPD. Patients with stable COPD (age 65.2±8.7 yrs (mean±SD), n=921) were enrolled in two identical randomized double-blind placebo-controlled 1-yr studies. Patients inhaled tiotropium 18 µg or placebo (mean screening forced expiratory volume in one second (FEV_1) 1.01 versus 0.99 L, 39.1 and 38.1% of the predicted value) once daily as a dry powder. The primary spirometric outcome was trough FEV_1 (i.e. FEV_1 prior to dosing). Changes in dyspnoea were measured using the Transition Dyspnea Index, and health status with the disease-specific St. George's Respiratory Questionnaire and the generic Short Form 36. Medication use and adverse events were recorded. Tiotropium provided significantly superior bronchodilation relative to placebo for trough FEV_1 response (approximately 12% over baseline) ($p<0.01$) and mean response during the 3 h following dosing (approximately 22% over baseline) ($p<0.001$) over the 12-month period. Tiotropium recipients showed less dyspnoea ($p<0.001$), superior health status scores, and fewer COPD exacerbations and hospitalizations ($p<0.05$). Adverse events were comparable with placebo, except for dry mouth incidence (tiotropium 16.0% versus placebo 2.7%, $p<0.05$). Tiotropium is an effective, once-daily bronchodilator that reduces dyspnoea and chronic obstructive pulmonary disease exacerbation frequency and improves health status. This suggests that tiotropium will make an important contribution to chronic obstructive pulmonary disease therapy.

A Novel Clinical Test of Respiratory Muscle Endurance—Hart N, Hawkins P, Hamnegard CH, Green M, Moxham J, Polkey MI. Eur Respir J 2002 Feb;19(2):232-239.

Impaired respiratory muscle endurance (RME) could reduce exercise tolerance and contribute to ventilatory failure. The aim of the present study was to develop a clinically-feasible method to measure RME using negative-pressure inspiratory-threshold loading. It was hypothesized that endurance time (tlim) could be predicted by normalizing oesophageal pressure-time product (PTP) per total breath cycle (PTP_{oes}) for maximum oesophageal pressure ($P_{oes,max}$); the load/capacity ratio. The corresponding mouth pressures, PTP_{mouth} and $P_{mouth,max}$ were also measured. The RME test was performed on 30 healthy subjects exposed to the same target pressure (70% of $P_{oes,max}$). Eight patients with systemic sclerosis/interstitial lung disease were studied to assess the validity and acceptability of the technique. Normal subjects showed a wide intersubject variation in tlim (coefficient of variation, 69%), with a linear relationship demonstrated between log tlim and $PTP_{oes}/P_{oes,max}$ ($r=0.88$). All patients with systemic sclerosis/interstitial lung disease had normal respiratory muscle strength, but six out of eight had a reduction in RME. In conclusion, endurance time can be predicted from the load/capacity ratio, over a range of breathing strategies; this relationship allows abnormal respiratory muscle endurance to be detected in patients. Oesophageal and mouth pressure showed a close correlation, thus suggesting that the test could be applied noninvasively.

Misuse of Corticosteroid Metered-Dose Inhaler Is Associated with Decreased Asthma Stability—Giraud V, Roche N. Eur Respir J 2002 Feb;19(2):246-251.

This study assessed whether the improper use of pressurized metered-dose inhalers (pMDIs) is associated with decreased asthma control in asthmatics treated by inhaled corticosteroids (ICS). General practitioners (GPs) included consecutive asthmatic outpatients treated by pMDI-administered ICS and on-demand, short-acting β_2-agonists. They measured an asthma instability score (AIS) based on daytime and nocturnal symptoms, exercise-induced dyspnoea, β_2-agonist usage, emergency-care visits and global perception of asthma control within the preceding month; the inhalation technique of the patient also was assessed. GPs (n=915) included 4,078 adult asthmatics; 3,955 questionnaires were evaluable. pMDI was misused by 71% of patients, of which 47% was due to poor coordination. Asthma was less stable in pMDI misusers than in good users (AIS: 3.93 versus 2.86, $p<0.001$). Among misusers, asthma was less stable in poor coordinators (AIS: 4.38 versus 3.56 in good coor-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dinators, p<0.001). To conclude, misuse of pressurized metered-dose inhalers, which is mainly due to poor coordination, is frequent and associated with poorer asthma control in inhaled corticosteroid-treated asthmatics. This study highlights the importance of evaluating inhalation technique and providing appropriate education in all patients, especially before increasing inhaled corticosteroid dosage or adding other agents. The use of devices which alleviate coordination problems should be reinforced in pressurized metered-dose inhaler misusers.

Noninvasive Ventilation in Cystic Fibrosis Patients with Acute or Chronic Respiratory Failure—Madden BP, Kariyawasam H, Siddiqi AJ, Machin A, Pryor JA, Hodson ME. Eur Respir J 2002 Feb;19(2):310-313.

The experience of using noninvasive ventilation (NIV) in 113 adult cystic fibrosis (CF) patients with chronic respiratory failure, during episodes of acute deterioration in respiratory function is reported. The patients aged 15-44 yrs were divided into three groups. Group A consisted of 65 patients (median forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) 0.7/1.4 L) who were on a lung transplant waiting list. Group B consisted of 25 patients (median FEV_1/FVC 0.7/1.4 L) who were being evaluated for lung transplantation. Group C consisted of 23 patients (median FEV_1/FVC 0.6/1.2 L) who were not being considered for lung transplantation. The mean duration of NIV support for groups A, B and C was 61 (range: 1-600) days, 53 (1-279) days and 45 (0.5-379) days respectively. Twenty-three patients in group A subsequently received lung transplantation and 12 of these patients had a median survival of 39 months postsurgery. Thirty-nine patients died and three awaited transplantation. Five patients in group B received a transplant four of whom survived; thirteen patients died and seven awaited transplantation. Twenty patients in group C died. Noninvasive ventilation improved hypoxia but failed to correct hypercapnia in these cystic fibrosis patients. Noninvasive ventilation is useful in the treatment of acute episodes of respiratory failure in cystic fibrosis patients with end-stage lung disease who have been accepted, or are being evaluated, for lung transplantation. For these patients, there is a possibility of prolonging life if they are successfully treated for their acute episode of respiratory failure until transplantation. In this group, treatment is not merely prolonging the process of dying.

Mild Therapeutic Hypothermia to Improve the Neurologic Outcome After Cardiac Arrest—The Hypothermia After Cardiac Arrest Study Group. N Engl J Med 2002 Feb 21;346(8):549-556.

BACKGROUND: Cardiac arrest with widespread cerebral ischemia frequently leads to severe neurologic impairment. We studied whether mild systemic hypothermia increases the rate of neurologic recovery after resuscitation from cardiac arrest due to ventricular fibrillation. METHODS: In this multicenter trial with blinded assessment of the outcome, patients who had been resuscitated after cardiac arrest due to ventricular fibrillation were randomly assigned to undergo therapeutic hypothermia (target temperature, 32 ° C to 34 ° C, measured in the bladder) over a period of 24 hours or to receive standard treatment with normothermia. The primary end point was a favorable neurologic outcome within six months after cardiac arrest; secondary end points were mortality within six months and the rate of complications within seven days. RESULTS: Seventy-five of the 136 patients in the hypothermia group for whom data were available (55 percent) had a favorable neurologic outcome (cerebral-performance category, 1 [good recovery] or 2 [moderate disability]), as compared with 54 of 137 (39 percent) in the normothermia group (risk ratio, 1.40; 95 percent confidence interval, 1.08 to 1.81). Mortality at six months was 41 percent in the hypothermia group (56 of 137 patients

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died), as compared with 55 percent in the normothermia group (76 of 138 patients; risk ratio, 0.74; 95 percent confidence interval, 0.58 to 0.95). The complication rate did not differ significantly between the two groups. CONCLUSIONS: In patients who have been successfully resuscitated after cardiac arrest due to ventricular fibrillation, therapeutic mild hypothermia increased the rate of a favorable neurologic outcome and reduced mortality.

Treatment of Comatose Survivors of Out-of-Hospital Cardiac Arrest with Induced Hypothermia—Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Gutteridge G, Smith K. N Engl J Med 2002 Feb 21;346(8):557-563.

BACKGROUND: Cardiac arrest outside the hospital is common and has a poor outcome. Studies in laboratory animals suggest that hypothermia induced shortly after the restoration of spontaneous circulation may improve neurologic outcome, but there have been no conclusive studies in humans. In a randomized, controlled trial, we compared the effects of moderate hypothermia and normothermia in patients who remained unconscious after resuscitation from out-of-hospital cardiac arrest. METHODS: The study subjects were 77 patients who were randomly assigned to treatment with hypothermia (with the core body temperature reduced to 33 ° C within 2 hours after the return of spontaneous circulation and maintained at that temperature for 12 hours) or normothermia. The primary outcome measure was survival to hospital discharge with sufficiently good neurologic function to be discharged to home or to a rehabilitation facility. RESULTS: The demographic characteristics of the patients were similar in the hypothermia and normothermia groups. Twenty-one of the 43 patients treated with hypothermia (49 %) survived and had a good outcome—that is, they were discharged home or to a rehabilitation facility—as compared with 9 of the 34 treated with normothermia (26 %, p=0.046). After adjustment for base-line differences in age and time from collapse to the return of spontaneous circulation, the odds ratio for a good outcome with hypothermia as compared with normothermia was 5.25 (95% confidence interval, 1.47 to 18.76; p=0.011). Hypothermia was associated with a lower cardiac index, higher systemic vascular resistance, and hyperglycemia. There was no difference in the frequency of adverse events. CONCLUSIONS: Our preliminary observations suggest that treatment with moderate hypothermia appears to improve outcomes in patients with coma after resuscitation from out-of-hospital cardiac arrest.

Change in Lung Function and Morbidity from Chronic Obstructive Pulmonary Disease in Alpha1-Antitrypsin MZ Heterozygotes: A Longitudinal Study of the General Population—Dahl M, Tybjarg-Hansen A, Lange P, Vestbo J, Nordestgaard BG. Ann Intern Med 2002 Feb 19;136(4):270-279.

BACKGROUND: A deteriorating effect of severe α_1-antitrypsin deficiency (ZZ genotype) on lung function is well known, whereas the role of intermediate deficiency (MZ genotype) remains uncertain. OBJECTIVE: To test the hypothesis that MZ intermediate α_1-antitrypsin deficiency affects pulmonary function and disease. DESIGN: Population-based cohort study with 21-year follow-up. SETTING: Copenhagen, Denmark. PARTICIPANTS: 9187 adults randomly selected from the Danish general population. MEASUREMENTS: Plasma α_1-antitrypsin levels, annual decrease in FEV_1, airway obstruction, and hospitalization and mortality from chronic obstructive pulmonary disease (COPD). RESULTS: 451 participants (4.9%) carried the MZ genotype. Plasma α_1-antitrypsin levels were 31% lower in MZ heterozygotes than in persons with the MM genotype (Student t -test, $p < 0.001$). Annual decrease in FEV_1 was 25 mL in MZ heterozygotes and 21 mL in persons with the MM genotype (t -test, $p = 0.048$). Airway obstruction was found in 19% of MZ heterozygotes compared with 15% of MM carriers (χ^2 test, $p = 0.023$); in a logistic regression analysis adjusted for age, sex, and tobacco consumption, the corresponding odds ratio was 1.3 (CI, 1.0 to 1.7). The incidence of hospitalization and mortality from COPD was 32 cases per 10 000 person-years in persons with the MZ genotype and 22 cases per 10 000 person-years in those with the MM genotype (log-rank test, $p = 0.063$). In a Cox regression model adjusted for age, sex, tobacco use, and FEV_1 at study entry, relative risk for COPD outcomes in persons with the MZ genotype versus persons with the MM genotype was 1.5 (CI, 1.0 to 2.3). All these results were independent of the S and E alleles in this gene and were not affected by cystic fibrosis Delta F508 heterozygosity. CONCLUSIONS: MZ heterozygotes had a slightly greater rate of decrease in FEV_1 and were modestly over-represented among persons with airway obstruction and COPD. In the population at large, MZ heterozygosity may account for a fraction of COPD cases—on the order of 2%, similar to the percentage of persons with COPD who have the severe but rare ZZ genotype.

Systematic Review of Randomised Controlled Trials of Over the Counter Cough Medicines for Acute Cough in Adults—Schroeder K, Fahey T. BMJ 2002 Feb 9;324(7333):329-331.

OBJECTIVES: To determine whether over the counter cough medicines are effective for acute cough in adults. DESIGN: Systematic review of randomised controlled trials. DATA SOURCES: Search of the Cochrane Acute Respiratory Infections Group specialised register, Cochrane Controlled Trials Register, Medline, Embase, and the UK Department of Health National Research Register in all languages. Included studies: All randomised controlled trials that compared oral over the counter cough preparations with placebo in adults with acute cough due to upper respiratory tract infection in ambulatory settings and that had cough symptoms as an outcome. RESULTS: 15 trials involving 2166 participants met all the inclusion criteria. Antihistamines seemed to be no better than placebo. There was conflicting evidence on the effectiveness of antitussives, expectorants, antihistamine-decongestant combinations, and other drug combinations compared with placebo. CONCLUSION: Over the counter cough medicines for acute cough cannot be recommended because there is no good evidence for their effectiveness. Even when trials had significant results, the effect sizes were small and of doubtful clinical relevance. Because of the small number of trials in each category, the results have to be interpreted cautiously.

Outcome After Cardiopulmonary Resuscitation in Patients with Pulmonary Arterial Hypertension—Hoeper MM, Galie N, Murali S, Olschewski H, Rubenfire M, Robbins IM, et al. Am J Respir Crit Care Med 2002 Feb 1;165(3):341-344.

Patients with pulmonary arterial hypertension (PAH) often die from right heart failure or sudden cardiac death. Cardiopulmonary resuscitation (CPR) may be instituted in these patients but there are no data in the medical literature about the outcome of CPR in this group of patients. We conducted a retrospective multicenter international study on the frequency and results of CPR in patients with PAH. A total of 3,130 patients with PAH were treated between 1997 and 2000 in 17 referral centers in Europe and in the United States. During this period, 513 patients had circulatory arrest and CPR was attempted in 132 (26%) of these patients. Although 96% of the CPR attempts took place in hospitalized patients (74% in intensive care units or equally equipped facilities) and although there was only minimal delay between collapse and initiation of CPR, resuscitation efforts were primarily unsuccessful in 104 patients (79%). Only eight patients (6%) survived for more than 90 d; these patients had no residual neurologic deficit. Hemodynamics obtained within 3 mo before CPR did not show any significant differences between the survivors and nonsurvivors. Except for one patient, all long-term survivors had identifiable causes of circulatory arrest that were rapidly reversible. Our data indicate that CPR for circulatory arrest in patients with PAH is rarely successful unless the cause of the cardiopulmonary decompensation can be corrected.

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Variable Tidal Volume Ventilation Improves Lung Mechanics and Gas Exchange in a Rodent Model of Acute Lung Injury—Arold SP, Mora R, Lutchen KR, Ingenito EP, Suki B. Am J Respir Crit Care Med 2002 Feb 1;165(3):366-371.

Random variations in breath rate and tidal volume during mechanical ventilation in the setting of acute lung injury have been shown to improve arterial oxygen tension. To test whether this improvement occurs over a specific range of variability, we examined several ventilation protocols in guinea pigs with endotoxin-induced lung injury. In Group I (n = 10), after 30 min of conventional volume-cycled ventilation, animals were ventilated with variable ventilation for 30-min intervals, during which time tidal volume was randomly varied by 10, 20, 40, and 60% of the mean, while simultaneously adjusting the frequency to maintain constant minute ventilation. In a second group of animals (Group II, n = 4), conventional volume-cycled ventilation was administered for 3 h. Variable ventilation significantly improved lung function over conventional volume-cycled ventilation. In Group I, lung elastance decreased, and blood oxygenation increased significantly during periods of 40 and 60% variable ventilation ($p < 0.05$) compared with conventional ventilation. These data indicate that variable ventilation is effective in improving lung function and gas exchange during acute lung injury.

The Impact of Follow-Up Physician Visits on Emergency Readmissions for Patients with Asthma and Chronic Obstructive Pulmonary Disease: A Population-Based Study—Sin DD, Bell NR, Svenson LW, Man SF. Am J Med 2002 Feb;112(2):120-125.

To examine the relation between follow-up office visits after emergency discharge and the risk of emergency readmissions in patients with asthma or chronic obstructive pulmonary disease (COPD).We used population-based data to identify all patients in Alberta, Canada, who had at least one emergency visit for asthma or COPD between April 1, 1996, and March 31, 1997 (N = 25 256). A Cox proportional hazards model was used to estimate the adjusted relative risk (RR) of a repeat visit to an emergency department within 90 days of an initial emergency visit in patients who did or did not have an office follow-up within the first 30 days.There were 7829 patients (31%) who had an office visit during the 30 days after their initial emergency encounter. Follow-up visits were associated with a significant reduction in the 90-day risk of an emergency readmission (RR = 0.79; 95% confidence interval [CI]: 0.73 to 0.86). Sensitivity analyses showed that a follow-up visit was inversely associated with a repeat emergency visit after adjusting for age, sex, area of residence, and income.Although these data should be interpreted with caution because of missing information on factors such as quality of care, they suggest that follow-up office visits are effective in reducing early relapses in patients who have been recently treated in emergency departments for asthma or COPD.

Anesthetic Care in Developing Countries: Equipment and Techniques—Tobias JD, Kim Y, Davis J. South Med J 2002 Feb;95(2):239-247.

Various organizations have been formed to provide short-term surgical services in developing countries. In these countries, the ability to provide effective anesthetic care requires the team to transport most of the equipment necessary to provide perioperative care for various surgical procedures. We review the basic supplies necessary to provide such care and discuss some of the issues related to the anesthesia machines, techniques, and monitoring in developing countries. Suggestions and options for preoperative assessment, intraoperative anesthetic care, and postoperative care, including pain management, are provided.

Endoscopic Ultrasound Guided Biopsy of Mediastinal Lesions Has a Major Impact on Patient Management—Larsen SS, Krasnik M, Vilmann P, Jacobsen GK, Pedersen JH, Faurschou P, Folke K. Thorax 2002 Feb;57(2):98-103.

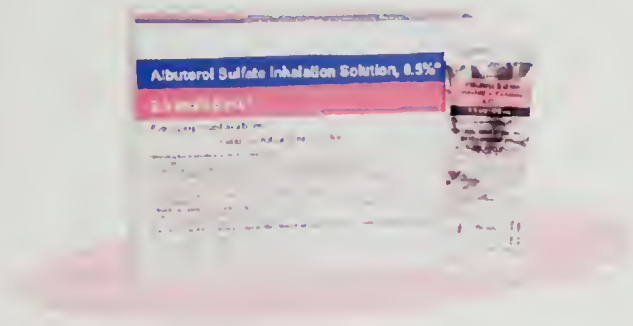
BACKGROUND: A study was undertaken to evaluate the clinical impact of endoscopic ultrasound guided fine needle aspiration biopsy (EUS-FNA) in patients with mediastinal masses suspected of malignancy. METHODS: From April 1993 to December 1999, 84 patients were referred for EUS-FNA. In all patients CT scanning had shown a lesion of the mediastinum suspected of malignancy located adjacent to the oesophagus. In order to evaluate the clinical impact of EUS-FNA, the history of each patient up to referral for EUS-FNA was reviewed. A board of thoracic specialists was asked to decide the further course of the patient if EUS-FNA had not been available, and this diagnostic strategy was compared with the actual clinical course after EUS-FNA. RESULTS: For the 79 patients in whom sufficient verification was obtained, EUS-FNA had a sensitivity of 92%, specificity of 100%, PPV of 100%, NPV of 80%, and an accuracy of 94% for cancer of the mediastinum. In 18 of 37 patients (49%) a thoracotomy/thoracoscopy was avoided as a result of EUS-FNA, and in 28 of 41 patients (68%) a mediastinoscopy was avoided. The direct result of the cytological diagnosis obtained by EUS-FNA was that a final diagnosis of small cell lung cancer was made in eight patients resulting in referral for chemotherapy, and in another three patients with benign disease specific treatment could be initiated (sarcoidosis, mediastinal abscess, and leiomyoma of the oesophagus). CONCLUSIONS: EUS-FNA is a safe and sensitive minimally invasive method for evaluating patients with a solid lesion of the mediastinum suspected by CT scanning. EUS-FNA has a significant impact on patient management and should be considered for diagnosing the spread of cancer to the mediastinum in patients with lung cancer considered for surgery, as well as for the primary diagnosis of solid lesions located in the mediastinum adjacent to the oesophagus.

Risk Factors for Onset and Remission of Atopy, Wheeze, and Airway Hyperresponsiveness—Xuan W, Marks GB, Toelle BG, Belousova E, Peat JK, Berry G, Woolcock AJ. Thorax 2002 Feb;57(2):104-109.

Background: Although many children with asthma may have a remission as they grow and other children who did not have asthma may develop asthma in adult life, knowledge about the factors that influence the onset and prognosis of asthma during adolescence and young adulthood is very limited. Methods: A cohort of 8-10 year old children (n=718) living in Belmont, New South Wales, Australia were surveyed six times at 2 yearly intervals from 1982 to 1992, and then again 5 years later in 1997. From this cohort, 498 subjects had between three and seven assessments and were included in the analysis. Atopy, airway hyperresponsiveness (AHR), and wheeze in the last 12 months were measured at each survey. Late onset, remission, and persistence were defined based on characteristics at the initial survey and the changes in characteristics at the follow up surveys. Results: The proportion of subjects with late onset atopy (13.7%) and wheeze (12.4%) was greater than the proportion with remission of atopy (3.2%) and wheeze (5.6%). Having atopy at age 8-12 years (OR 2.8, 95% CI 1.5 to 5.1) and having a parental history of asthma (OR 2.0, 95% CI 1.02 to 4.13) were significant risk factors for the onset of wheeze. Having AHR at age 8-12 years was a significant risk factor for the persistence of wheeze (OR 4.3, 95% CI 1.3 to 15.0). Female sex (OR 1.9, 95% CI 1.01 to 3.60) was a significant risk factor for late onset AHR whereas male sex (OR 1.9, 95% CI 1.1 to 2.8) was a significant risk factor for late onset atopy. Conclusions: The onset of AHR is uncommon during adolescence, but the risk of acquiring atopy and recent wheeze for the first time continues during this period. Atopy, particularly present at the age of 8-10 years, predicts the subsequent onset of wheeze.

Sahaja Yoga in the Management of Moderate to Severe Asthma: A Randomised Controlled Trial—Manocha R, Marks GB, Kenchington P, Peters D, Salome CM. Thorax 2002 Feb;57(2):110-115.

BACKGROUND: Sahaja Yoga is a traditional system of meditation based on yogic principles which may be used for therapeutic purposes. A



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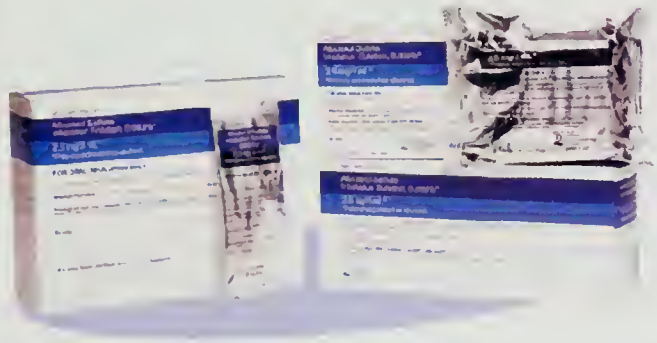

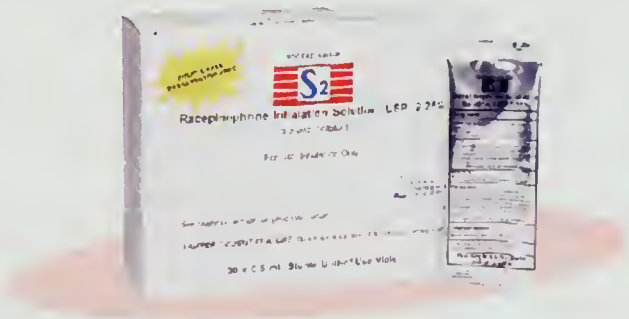

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study was undertaken to assess the effectiveness of this therapy as an adjunctive tool in the management of asthma in adult patients who remained symptomatic on moderate to high doses of inhaled steroids. METHODS: A parallel group, double blind, randomised controlled trial was conducted. Subjects were randomly allocated to Sahaja yoga and control intervention groups. Both the yoga and the control interventions required the subjects to attend a 2 hour session once a week for 4 months. Asthma related quality of life (AQLQ, range 0-4), Profile of Mood States (POMS), level of airway hyperresponsiveness to methacholine (AHR), and a diary card based combined asthma score (CAS, range 0-12) reflecting symptoms, bronchodilator usage, and peak expiratory flow rates were measured at the end of the treatment period and again 2 months later. RESULTS: Twenty one of 30 subjects randomised to the yoga intervention and 26 of 29 subjects randomised to the control group were available for assessment at the end of treatment. The improvement in AHR at the end of treatment was 1.5 doubling doses (95% confidence interval (CI) 0.0 to 2.9, p=0.047) greater in the yoga intervention group than in the control group. Differences in AQLQ score (0.41, 95% CI -0.04 to 0.86) and CAS (0.9, 95% CI -0.9 to 2.7) were not significant (p>0.05). The AQLQ mood subscale did improve more in the yoga group than in the control group (difference 0.63, 95% CI 0.06 to 1.20), as did the summary POMS score (difference 18.4, 95% CI 0.2 to 36.5, p=0.05). There were no significant differences between the two groups at the 2 month follow up assessment. CONCLUSIONS: This randomised controlled trial has shown that the practice of Sahaja yoga does have limited beneficial effects on some objective and subjective measures of the impact of asthma. Further work is required to understand the mechanism underlying the observed effects and to establish whether elements of this intervention may be clinically valuable in patients with severe asthma.

A Novel Hypothesis to Explain the Bronchconstrictor Effect of Deep Inspiration in Asthma—Burns GP, Gibson GJ. Thorax 2002 Feb;57(2):116-119.

Background: In healthy subjects deep inspiration transiently dilates the airways, while many asthmatic subjects show bronchoconstriction by a mechanism which is incompletely understood. We hypothesised that the negative intrathoracic pressure associated with deep inspiration occurring in the context of increased leakiness of the airway vasculature may temporarily increase airway oedema and thus reduce luminal diameter in subjects with asthma. Methods: The effects of non-forced deep inspiration and forced deep inspiration through resistance (generating enhanced negative intrathoracic pressure) on specific airway conductance (sG_{aw}) were compared in 10 asthmatic and 11 healthy subjects. Each performed two respiratory manoeuvres: (1) sG_{aw} was measured immediately after three deep inspirations without added resistance, each performed at a predetermined rate (equal to that when performed maximally through resistance); and (2) sG_{aw} was measured immediately after three forced inspirations through resistance. Results: Compared with deep inspiration without added resistance, sG_{aw} was significantly reduced after deep inspiration through resistance in the asthmatic group by a mean (SD) of -13.5 (11.0)% (p=0.003) but was unchanged in the control group (-0.5 (12.4)%, p=0.67). Conclusions: Given the similar time-volume relationship in the two manoeuvres, the reduction in sG_{aw} is unlikely to be due to differences in smooth muscle behaviour. It is suggested that the enhanced negative intrathoracic pressure acting across the airway capillaries increases airway wall oedema and reduces airway calibre. Such a mechanism operational during normal rapid deep inspiration might contribute to bronchoconstriction after deep inspiration in subjects with asthma.

Relaxation Therapies for Asthma: A Systematic Review—Huntley A, White AR, Ernst E. Thorax 2002 Feb;57(2):127-131.

Background: Emotional stress can either precipitate or exacerbate both acute and chronic asthma. There is a large body of literature available on the use of relaxation techniques for the treatment of asthma symptoms. The aim of this systematic review was to determine if there is any evidence for or against the clinical efficacy of such interventions. Methods: Four independent literature searches were performed on MEDLINE, Cochrane Library, CISCOM, and Embase. Only randomised clinical trials (RCTs) were included. There were no restrictions on the language of publication. The data from trials that statistically compared the treatment group with that of the control were extracted in a standardised predefined manner and assessed critically by two independent reviewers. Results: Fifteen trials were identified, of which nine compared the treatment group with the control group appropriately. Five RCTs tested progressive muscle relaxation or mental and muscular relaxation, two of which showed significant effects of therapy. One RCT investigating hypnotherapy, one of autogenic training, and two of biofeedback techniques revealed no therapeutic effects. Overall, the methodological quality of the studies was poor. Conclusions: There is a lack of evidence for the efficacy of relaxation therapies in the management of asthma. This deficiency is due to the poor methodology of the studies as well as the inherent problems of conducting such trials. There is some evidence that muscular relaxation improves lung function of patients with asthma but no evidence for any other relaxation technique.

Expiratory Muscle Endurance in Chronic Obstructive Pulmonary Disease—Ramirez-Sarmiento A, Orozco-Levi M, Barreiro E, Mendez R, Ferrer A, Broquetas J, Gea J. Thorax 2002 Feb;57(2):132-136.

Background: A reduction in expiratory muscle (ExM) endurance in patients with chronic obstructive pulmonary disease (COPD) may have clinically relevant implications. This study was carried out to evaluate ExM endurance in patients with COPD. Methods: Twenty three patients with COPD (FEV_1 35 (14)% predicted) and 14 matched controls were studied. ExM endurance was assessed using a method based on the use of an expiratory threshold valve which includes two steps. In step 1 the load is progressively increased (50 g every 2 minutes) until task failure is reached, and the pressure generated against the highest tolerated load is defined as the maximal expiratory sustainable pressure (Pth_{max}). In step 2 subjects breathe against a submaximal constant load (80% of Pth_{max}) and the time elapsed until task failure is termed the expiratory endurance time (Tth_{80}). In addition, the strength of peripheral muscles (handgrip, HGS) and respiratory muscles (maximal inspiratory and expiratory pressures, PI_{max} and PE_{max}, respectively) was evaluated. Results: Patients with COPD had lower ExM strength and endurance than controls: PE_{max} 64 (19)% predicted v 84 (14)% predicted (mean difference 20%; 95% confidence intervals (CI) 14 to 39); Pth_{max} 52 (27) v 151 (46) cm H_2O (mean difference 99, 95% CI 74 to 123); and Tth_{80} 9.4 (6.3) v 14.2 (7.4) min (mean difference 4.8, 95% CI 1.0 to 10.4; p<0.01 for all). Interestingly, ExM endurance directly correlated with both the severity of airways obstruction (Pth_{max} with FEV_1, r=0.794, p<0.01) and the reduction in strength observed in different muscle groups (Pth_{max} with HG, PI_{max} or PE_{max}, r=0.550, p<0.05; r=0.583, p<0.001; and r=0.584, p<0.001, respectively). Conclusions: ExM endurance is decreased in patients with COPD. This impairment is proportional to the severity of the disease and is associated with lower strength in different muscle groups. This suggests that systemic effects are implicated in the impairment observed in ExM function.

Clinical Audit Indicators of Outcome Following Admission to Hospital with Acute Exacerbation of Chronic Obstructive Pulmonary Disease—Roberts CM, Lowe D, Bucknall CE, Ryland I, Kelly Y, Pearson MG. Thorax 2002 Feb;57(2):137-141.

Background: The 1997 BTS/RCP national audit of acute chronic obstructive pulmonary disease (COPD) in terms of process of care has previously been reported. This paper describes from the same cases the outcomes of death, readmission rates within 3 months of initial admission, and length of stay. Identification of the main pre-admission predictors of outcome may be used to control for confounding factors in population characteristics when comparing performance between units. Methods: Data on 74 variables were collected retrospectively using an audit proforma from patients admitted to UK hospitals with acute COPD. Important prognostic variables for the three outcome measures were identified by relative risk and logistic regression was used to place these in order of predictive value. Results: 1400 admissions from 38 acute hospitals were collated. 14% of cases died within 3 months of admission with variation between hospitals of 0-50%. Poor performance status, acidosis, and the presence of leg oedema were the best significant independent predictors of death. Age above 65, poor performance status, and lowest forced expiratory volume in 1 second (FEV_1) tertile were the best predictors of length of stay (median 8 days). 34% of patients were readmitted (range 5-65%); lowest FEV_1 tertile, previous admission, and readmission with five or more medications were the best predictors for readmission. Conclusions: Important predictors of outcome have been identified and formal recording of these may assist in accounting for confounding patient characteristics when making comparisons between hospitals. There is still wide variation in outcome between hospitals that remains unexplained by these factors. While some of this variance may be explained by incomplete recording of data or patient factors as yet unidentified, it seems likely that deficiencies in the process of care previously identified are responsible for poor outcomes in some units.

Patients' and Carers' Preferences in Two Models of Care for Acute Exacerbations of COPD: Results of a Randomised Controlled Trial—Ojoo JC, Moon T, McGlone S, Martin K, Gardiner ED, Greenstone MA, Morice AH. Thorax 2002 Feb;57(2):167-169.

BACKGROUND: Patients with an acute exacerbation of chronic obstructive pulmonary disease (COPD) were randomised to either hospital at home (HaH) or inpatient management, and patient and carer preferred site of management and satisfaction with care received in the two arms was determined. METHODS: Emergency admissions with an acute exacerbation of COPD were randomised to inpatient care or HaH care. After discharge an independent observer administered a questionnaire to both patients and carers on the preferred site of care and scored satisfaction with the care received. RESULTS: Of 60 patients recruited, 30 were randomised to receive HaH care. Retrospective patient preference for HaH care was 96.3% in the domiciliary arm and 59.3% in the conventional arm; carer preference figures were 85.7% and 42.9%, respectively. There was a higher preference for domiciliary care by both patients and carers in the HaH arm than in the inpatient arm (p=0.001 and p=0.01, respectively). Patients recorded equal satisfaction with care in the two arms (88.1% in the conventional arm, 91.7% in the domiciliary arm); carer scores were 91.3% and 91.9%, respectively. CONCLUSIONS: The results of this study show that both patients and carers were significantly more likely to prefer domiciliary care if they were in the HaH arm. Since patients had to be willing to be looked after at home, both patients' and carers' perceptions of the benefits of HaH care were reinforced by their experience. HaH care of acute exacerbations of COPD is the preferred option in suitable patients.

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The Pulmonary Physician in Critical Care # 2: Oxygen Delivery and Consumption in the Critically Ill—Leach RM, Treacher DF. Thorax 2002 Feb;57(2):170-177.

Early detection and correction of tissue hypoxia is essential if progressive organ dysfunction and death are to be avoided. However, hypoxia in individual tissues or organs caused by disordered regional distribution of oxygen delivery or disruption of the processes of cellular oxygen uptake and utilisation cannot be identified from global measurements. Regional oxygen transport and cellular utilisation have an important role in maintaining tissue function. When tissue hypoxia is recognised, treatment must be aimed at the primary cause. Supplemental oxygen may be life saving in some situations but cannot correct inadequate oxygen delivery caused by a low cardiac output or impaired ventilation. Recent innovations include artificial oxygen carrying proteins and "haemoglobin" molecules designed to improve tissue blood flow by reducing viscosity. Regulating cell metabolism using different substrates or drugs has so far been poorly explored but is an exciting area for further research. A minimum level of global oxygen delivery and perfusion pressure must be maintained in the critically ill patient with established "shock", but advances in the understanding and control of regional distribution and other "downstream" factors in the oxygen cascade are needed to improve outcome in these patients.

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Gordon D. Rubenfeld, MD and Richard D. Branson, BA, RRT, FAARC

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Mark J. Heulitt, MD, FAARC, and Richard D.Branson, BA, RRT, FAARC

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Jon D. Nilsestuen, PhD, RRT, FAARC and Richard D. Branson, BA, RRT, FAARC

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PROGRAM 5: Talking With Patients and Families About Death and Dying

Helen M. Sorensen, MA, RRT, FAARC and Dovid J. Pierson, MD, FAARC

Learn how communication is a key to easing suffering at end of life and how to make dying easily understood by all parties. Also, learn how attitudes toward death vary among different ethnic groups and how to recognize the importance these differences play in termination of life support.

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PROGEAM 6: Pressure versus Volume Ventilation: Does It Matter?

Robert S. Compbell, RRT, FAARC and Richard D. Branson, BA, RRT, FAARC

This program will give you a grasp of all the variables that enter into the decision to use pressure or volume ventilation. You will gain an understanding of the physiology of assisted breathing and how it is key to final selection of ventilatory mode. You will also learn what is displayed on ventilator screens and where to look for the information.

Live Videoconference September 10, 11:30 a.m. – 1:00 p.m. Central Time
Teleconference with Video October 8, 11:30 a.m. – 12:00 Noon Central Time

PROGRAM 7: Inpatient Management of COPD

Randall Rosenblatt, MD and David J. Pierson, MD, FAARC

Learn the accepted guidelines for identifying and treating patients with COPD, including the therapeutic efficacy of steroids in the management of the acute exacerbation of COPD. You will also learn about respiratory failure and other potential complications that would require intervention in the patient's care.

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PROGRAM 8: High-Frequency Oscillatory Ventilation

Thomas E. Stewart, MD and Richard D. Branson, BA, RRT, FAARC

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Educating Respiratory Care Professionals: An Emphasis on Critical Thinking

It is not surprising that educators in any discipline desire to improve their students' thinking abilities to help them make better personal and professional decisions. The emphasis on critical thinking is nothing new and can be traced back to ancient times and accounts of how students were challenged to think about their knowledge, beliefs, and behaviors. For example, it is widely known that Socrates would press his students until they could provide reasons and evidence to support their arguments. He would dismiss those beliefs and decisions that could not be supported, causing dismay among his students and unrest among the politicians. The Socratic method has survived the test of time and has become an important educational tool to prepare doctors, lawyers, educators, respiratory therapists, and a variety of other professionals.

See the Original Study on Page 571

The difficulties in describing and improving critical thinking are well documented in numerous definitions, theories, and educational practices. Developing critical thinking in respiratory therapy students has been proposed as a method for improving clinical decision-making, based on the hypothesis and preliminary data that a positive relationship exists between decision-making and critical thinking.[1–3] This issue of Respiratory Care provides additional evidence that there is a relationship between general critical thinking and decision-making in respiratory care.[4] Dr Thomas V Hill's study supports the belief that students with strong critical thinking abilities make better clinical decisions, using the Watson Glaser Critical Thinking Appraisal (WGCTA, Psychological Corporation, San Antonio, Texas) to measure critical thinking and the Clinical Simulation Self Assessment Examinations (CSSAE, developed by the National Board for Respiratory Care) to measure decision-making. In addition, the study describes current educational practices in respiratory care, reporting the predominant use of role modeling, case studies, clinical simulation, computer-assisted instruction, and small group discussion.[4]

Dr Hill's study also illustrates common difficulties in educational research and social sciences research in general, which frequently result in small sample sizes and the use of nonexperimental designs, since randomization is often not possible. Therefore, although we have enough data to document that a positive relationship exists between critical thinking and decision-making, we cannot determine cause and effect.[2–4] We are left to ponder which educational methods can improve our students' decision-making and critical thinking, although there are some data that support the use of problem-based learning in respiratory care.[5,6] I cannot help but smile and believe that Socrates would look favorably on the successful uses of problem-based learning,[7–10] while reminding us we need additional evidence to support its use in respiratory care.

Since most respiratory therapy programs are struggling with limited pools of qualified students, I would caution against Dr Hill's suggestion that assessment of critical thinking proficiency could be used as part of a process for evaluating prospective students.[4] I do not think we have the luxury at present of imposing additional acceptance criteria for admissions into our programs. However, I think his suggestion that we consider using the WGCTA to assess prospective faculty is an interesting proposition and deserves additional consideration.[4] I also believe that we can do a lot more to promote critical thinking among our practicing respiratory therapists. For example, the issues on Evidence-Based Medicine in Respiratory Care could be incorporated into continuing education and in-service programs to facilitate critical thinking and an evidence-based approach to practice among respiratory therapists.[11,12] We should also investigate which educational methods can improve the critical thinking of our practicing respiratory therapists.

In conclusion, we need further study of the effectiveness of educational strategies to promote the development of critical thinking and decision-making. As a means to evaluate program effectiveness, I encourage educational programs to use the WGCTA to assess their students' abilities on admission and prior to graduation to determine whether there are any changes in their students' critical thinking.[5] To those conducting educational research, I challenge you in the spirit of Socrates to work with other colleagues to design and implement a prospective, randomized study with sufficient power to add to our knowledge base of

those strategies that can improve critical thinking and decision-making in respiratory care.

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REFERENCES

1. Mishoe SC. Critical thinking, educational preparation, and development of respiratory care practitioners. Distinguished Papers Monograph 1993;2:29–43.
2. Mishoe SC, Dennison FH, Thomas-Goodfellow L. A comparison of respiratory therapy students' critical thinking abilities with performance on the clinical simulation examinations (abstract). Respir Care 1997;42(11):1078.
3. Shelledy DC, Valley MA, Murphy DE, Carpenter ME. Effects of content, process computer-assisted instruction, and critical thinking abilities on students' performance on written clinical simulations. Respir Care Educ Annu 1997;6:11–29.
4. Hill TV. The relationship between critical thinking and decision making in respiratory care students. Respir Care 2002;47(5):571-577.
5. Mishoe SC, Dennison FH, Thomas-Goodfellow L. Can respiratory therapy education improve critical thinking? (abstract) Respir Care 1997;42(11):1078.
6. O'pt Holt TB. A first year experience with problem-based learning in a baccalaureate cardiorespiratory care program. Respir Care Educ Annu 2000;9:47–58.
7. Benjamin EM, Schneider MS, Hinchey KT. Implementing practice guidelines for diabetes care using problem-based learning: a prospective controlled trial using firm systems. Diabetes Care 1999; 22(10):1672–1678.
8. Teshima DY. Outcome measurement of problem-based learning. Clin Lab Sci 2001;14:68–69.
9. Curtis JA, Indyk D, Taylor B. Successful use of problem-based learning in a third-year pediatric clerkship. Ambulatory Pediatr 2001; 1(3):132–135.
10. Antepohl W, Herzig S. Problem-based learning versus lecture-based learning in a course of basic pharmacology: a controlled randomized study. Med Educ 1999;33(2):106–113.
11. Conference on evidence-based medicine in respiratory care. Part I. Respir Care 2001;46(11):1200–1303.
12. Conference on evidence-based medicine in respiratory care. Part II. Respir Care 2001;46(12):1368–1449.

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Take a Deep Breath, Then *Blast* It Out

Dr Stoller and I used to think that spirometry was as easy as measuring the other vital signs. However, during the last couple of decades we've needed to rely on forced expiratory volume in the first second (FEV_1) values to determine the efficacy of dangerous or expensive medications for asthma and chronic obstructive pulmonary disease, and we've learned the hard way to distrust changes in FEV_1 until we've viewed the spirograms and flow-volume curves from each spirometry test session (before and after the intervention). Dr Stoller's article in this issue describes the incredible improvement in the quality of bedside spirometry done by respiratory therapists in his hospital after training by pulmonary function laboratory technicians of The Cleveland Clinic Foundation.[1]

See the Original Study on Page 578

Inaccurate spirometers are finally a rarity: nowadays poorly trained, timid, or harried coaches is the major reason for the high false positive rate of spirometry testing done outside hospital-based pulmonary function labs. Just before Thanksgiving of 2001 I reviewed the results of 1,500 spirometry tests done by respiratory therapists who volunteered to test New York police officers who had responded to the World Trade Center disaster. My confidence in the results would have been much higher if those therapists had first been trained by experienced pulmonary function technicians.

I've learned from Drs Allan Palmer and Alan Coates that the priority for coaching should be: (1) look at the patient and encourage a *deep* breath; (2) yell "blast out!" and (3) quietly tell the patient to keep on blowing out, while watching the spirometer for feedback. When the new National Lung Health Education Program[2,3] (NLHEP) office spirometers become commercially available, clinicians will save time and effort during phase 3 of that maneuver by stopping after only 6 seconds. These new spirometers will substitute the FEV_6 for the forced vital capacity, using Dr John Hankinson's National Health and Nutrition Examination Survey (NHANES III) equations for predicted values.[4]

At the 47th International Respiratory Congress in San Antonio in December of 2001, I presented a "rogue's gallery" of poor quality spirometry at Dr Thomas Petty's NLHEP symposium. I believe that if respiratory therapists learn how to perform good quality spirometry, not only will bedside spirometry become more accurate at detecting airway obstruction and improvements following bronchodilator treatments, they will then be able to offer spirometry training to the office staff of local primary care practitioners, who care for most of the community patients with asthma and chronic obstructive pulmonary disease—ideal clients to provide referrals to home care services, asthma management programs, and pulmonary rehabilitation programs run by respiratory therapists.

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REFERENCES

1. Stoller JK, Orens D, Hoisington E, McCarthy K. Bedside spirometry in a tertiary care hospital: The Cleveland Clinic Experience. Respir Care 2002;47(5):578-582.
2. Information on the National Lung Health Education Program and spirometry available at http://www.nlhep.org/spirom1.html (accessed Mar 4, 2002).
3. Ferguson GT, Enright PL, Buist AS, Higgins MW. Office spirometry for lung health assessment in adults: a consensus statement from the National Lung Health Education Program. Chest. 2000;117(4):1146–1161. Respir Care. 2000;45(5):513–530.
4. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. Am J Respir Crit Care Med 1999;159(1):179–187.

The Relationship Between Critical Thinking and Decision-Making in Respiratory Care Students

Thomas V Hill PhD RRT

INTRODUCTION: Respiratory therapists evaluate the condition of their patients, make decisions about the appropriateness of the treatment plan, provide care, and evaluate the outcomes of their decisions. To practice effectively they must think critically and make appropriate decisions. As educators make curriculum changes to improve students' decision-making (DM) skills, strategies to develop critical thinking (CT) have been proposed as one method of achieving that goal. This approach is based on the hypothesis that a positive relationship exists between DM and CT. The present study examines the relationship between CT and DM in respiratory care students. If there is a positive relationship between CT and DM, respiratory care training programs might improve the DM skills of future practitioners by providing instruction that improves CT. METHODS: Using a correlational research design, DM scores from the Clinical Simulation Self-Assessment Examination (CSSAE) were compared with scores from the Watson-Glaser Critical Thinking Appraisal for 143 graduating respiratory care students from 10 programs. Program directors completed a questionnaire and were then interviewed about the methods they employ to develop CT and DM in their students. RESULTS: A significant correlation between CT and DM was found for 110 students completing the Secure Form of the CSSAE ($r = 0.32$, $p < 0.01$). All programs used case studies and clinical simulations, and most used role modeling, computer-assisted instruction, patient rounds, small group discussion, and study skills courses. No differences were identified in the use of the CT and DM development strategies by different programs. CONCLUSIONS: The findings support the belief that students with strong CT proficiency make better clinical decisions. Assessment of CT proficiency could be used as one part of a process for evaluating prospective students and faculty for respiratory care programs. Program directors and faculty felt that case studies and clinical simulations are important strategies. *Key words: critical thinking, decision-making, respiratory therapists, training, education.* [Respir Care 2002;47(5):571–577]

Introduction

The practice of respiratory care has changed tremendously during its 50-year development. When the first inhalation technician positions were created in the 1940s, those practitioners were primarily responsible for delivering oxygen cylinders to the patient's bedside, where a physician or nurse would administer the oxygen to the patient by tent or mask. Since that time the profession has evolved and respiratory therapists now assume much more responsibility for initiating, monitoring, and modifying the care they provide. With the introduction of therapist-driven protocols, respiratory therapists have been empowered to independently evaluate the patient's condition and make decisions regarding the need for treatment. That level of practice requires the practitioner to process a great deal of information about the patient's condition, to make decisions about which treatments to initiate, to assess the ef-

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fectiveness of the treatments, and to implement necessary changes to the treatment plan. To effectively complete that process, practitioners must possess the proficiency to think critically and to make appropriate decisions.[1]

As the practice of respiratory care evolved, the techniques for training prospective practitioners also developed. Early training programs were based in hospitals, and the length of training ranged from several weeks to a year. Most teachers were physicians and practitioners with experience in practice but not necessarily a foundation in education. Currently most training occurs in colleges and universities, within formally accredited programs of 1–4 years. Educators today are well trained not only in the techniques for delivering patient care, but also in the theories of instructional planning, delivery, and evaluation.

Respiratory care programs have become quite proficient in teaching future practitioners appropriate command of the knowledge and technical skills necessary to function in the current clinical setting. Yet as health care becomes more concerned with documentation of quality outcomes, cost containment, and the effectiveness of care, respiratory therapists must be able to do more than follow the physician's instructions. They must be able to independently evaluate the patient's condition, make decisions about how to treat the patient, implement the treatment plan, and evaluate the outcomes of their decisions. Participants in a National Consensus Conference on Respiratory Care Education gave high rankings to the ability of future practitioners to analyze, assess, and problem-solve.[2] Educational programs are thus charged with teaching and evaluating students' ability to make appropriate decisions.

Ennis defined critical thinking (CT) as reasonable, reflective thinking focused on what to believe or do.[3] As educational programs make curriculum changes to improve students' decision-making (DM) skills, the introduction of processes to develop CT proficiency has been proposed as one method of achieving that goal. The rationale for focusing on improving DM by teaching CT is based on the hypothesis that a positive relationship exists between DM and CT. This relationship has been described in both nursing and respiratory care students,[4–6] but warrants further study.

The traditional lecture mode of instruction employed by many programs presents obstacles to the development of the student's CT proficiency.[7] In the lecture format, students are presented with information and concepts and then left on their own to analyze, prioritize, and structure their new knowledge. Lecture is a generally passive style of education for the students, in which CT is not taught. Programs face the challenge of teaching the vast amounts of information and concepts deemed necessary for competent practice while also developing the student's CT proficiency, sometimes within the short time frame of a 2-year program.

Several strategies have been proposed for developing CT in students, including achieving a balance between lecture and interaction, increasing student participation in learning, introducing students to poorly-structured problems within their discipline, and employing problem-based learning.[1,7–9] Though educational programs have experimented with some of those strategies to improve CT, their effectiveness has not been widely reported. A description of strategies used by programs whose students score higher in CT would benefit educators as they seek to improve these important abilities in their students.

The nature of the relationship between CT and DM has been the subject of much discussion and has been investigated by several researchers. Ennis's definition implies that the individual uses the process of CT to arrive at an appropriate decision.[3] A consensus definition from the American Philosophical Association focuses on CT as a process of purposeful judgment that places emphasis on DM in the context of an identified problem, goal, desired outcome, or purpose.[10] This notion of CT as the process to the outcome of DM is supported by others. Brookfield claims that if the individual thinks critically he or she will make good clinical decisions that are grounded in an accurate understanding of contexts for practice.[11] Case claims that CT improves the quality of professional and personal decisions.[12] In a proposed model of CT for nursing judgment, discipline-specific clinical DM is defined as the outcome of CT.[13]

Several studies have attempted to test for the existence of a relationship between CT and DM. Earlier studies found no significant correlation between CT and DM,[14–17] but later studies found a significant positive correlation. Mishoe et al[5] and Shelledy et al[6] reported significant relationships between CT and DM in respiratory care students and suggested that additional research is needed on the nature of the relationship between CT and DM.[1,6]

The present study examines the relationship between DM and CT in respiratory care students and describes strategies used by respiratory care training programs to develop CT and DM. The research questions include: (1) Do statistically significant relationships exist between CT and DM in respiratory care students? and (2) What methods do respiratory care training programs use to develop CT and DM in their students?

Methods

The first phase of the present study employed a correlational research procedure to describe the relationship between CT and DM. The sample consisted of graduating students enrolled in advanced practitioner respiratory care programs in a tri-state area, as indicated in the current list of accredited programs provided by the Committee on Accreditation for Respiratory Care. Initially, students from

Table 1. Demographic Data on Student Participants

Program	*n*	Age (y)*	Gender (F/M)	Years of College Prior to Entering Program*	Months of Employment Experience in Respiratory Care*	Grade Point Average*
1	15	30.7 ± 8.4	13/2	2.8 ± 0.9	4.7 ± 4.5	3.61 ± 0.30
2	10	28.9 ± 10.1	8/2	2.4 ± 2.1	5.6 ± 3.8	3.28 ± 0.23
3	22	30.6 ± 8.8	14/8	2.5 ± 1.4	7.2 ± 17.4	3.33 ± 0.44
4	5	26.0 ± 1.6	3/2	3.2 ± 1.6	17.6 ± 5.1	3.04 ± 0.27
5	21	31.2 ± 7.8	16/5	2.5 ± 1.8	4.1 ± 3.6	3.12 ± 0.45
6	18	27.1 ± 5.4	13/5	1.7 ± 1.0	7.3 ± 4.6	3.11 ± 0.39
7	9	25.8 ± 6.1	7/2	2.1 ± 1.6	11.8 ± 4.9	3.21 ± 0.30
8	18	29.6 ± 7.6	14/4	2.2 ± 1.3	7.8 ± 4.5	3.20 ± 0.43
9	18	23.2 ± 2.3	11/7	1.3 ± 1.1	7.5 ± 9.4	3.20 ± 0.38
10	7	24.6 ± 5.4	7/0	1.6 ± 0.6	4.0 ± 5.3	3.05 ± 0.23
All Students	143	28.3 ± 7.5	106/37	2.1 ± 1.4	7.0 ± 8.7	3.24 ± 0.40

* Values are mean ± SD.

29 programs were identified and invited to participate in the study through their program directors.

A letter was sent to the director of each respiratory care program in the tri-state area, inviting his or her program to participate in the study. The letter described the purpose of the study, requirements of programs and students who choose to participate, and a description of the benefits of participation. Each program director who chose to participate was asked to complete and return a program information form and a questionnaire about the strategies employed by his or her program to develop CT and DM in students. The questionnaire included teaching strategies that were identified in the literature, and additional space was provided to allow the program directors to add methods not previously identified. Before distribution to the program directors in the sample, the questionnaire was pilot-tested with 5 program directors from outside the geographic area of the study.

Following receipt of the completed questionnaire, a telephone interview was conducted with the program directors to obtain additional information about the educational methods employed by the programs to develop CT and DM. Their responses were transcribed exactly as provided and were analyzed using a constant comparison strategy.[18,19] Each item in the notes was coded, allowing comparison of the use of strategies by each program. As all of the responses were coded, differences and similarities were constantly compared until any apparent patterns of use or themes were identified. Similarities in the use of a particular strategy by at least half of the programs using that strategy were considered important and are described for each strategy.

The students were asked to provide demographic data, including years of college prior to entering the respiratory care program, months of employment experience in respiratory care (excluding educational clinical experience), and college grade point average (GPA).

The student's score on the Watson-Glaser Critical Thinking Appraisal (WGCTA, Psychological Corporation, San Antonio, Texas) measured CT. The WGCTA is a standardized, 80-item, multiple-choice test designed to assess CT abilities through reading comprehension. The total score on the WGCTA was used as a measure of CT proficiency, because the sub-scores are based on a relatively small number of items and may lack sufficient reliability when used in place of the total score.[20] The student's DM score on the Clinical Simulation Self-Assessment Examination (CSSAE, developed by the National Board for Respiratory Care) measured DM.[21] The WGCTA and CSSAE were administered by the program directors and/or faculty, using written instructions provided by the test authors. Statistical analyses were performed with commercially available statistical software (SPSS for Windows, Version 10.0.7, SPSS Inc, Chicago, Illinois).

After the WGCTA forms were returned to the investigator and scored, each program director received a report containing the CT scores of the students in his or her program, along with the corresponding percentile rank for subjects in the program's institutional classification. Each student received a report of his or her individual score and corresponding percentile rank.

Results

Results are reported for 143 students in the 10 participating programs that provided complete data for their students and participated in the telephone interviews. Students graduating from Program 4 receive a baccalaureate degree, whereas students graduating from the other 9 programs receive an associate degree.

Table 2. Results of Watson-Glaser Critical Thinking Appraisal

Program	Mean Score	Standard Deviation	Range
1	50.6	8.9	32–65
2	53.5	6.1	45–62
3	51.1	9.5	38–75
4	62.8	5.0	56–70
5	54.3	11.2	33–74
6	52.6	6.6	40–66
7	56.0	8.3	42–67
8	53.2	8.5	38–67
9	48.9	7.2	38–68
10	47.0	6.8	33–53
All Students	52.6	8.8	32–75

Table 3. Decision-Making Scores from the Clinical Simulation Self-Assessment Examination, Ranked by Score

CSSAE Form	Program	Decision-Making Scores*
Secure Form	4	103.4 ± 17.9
	8	99.1 ± 16.1
	1†	96.9 ± 22.6
	7	88.1 ± 28.4
	3	86.3 ± 18.5
	9	79.9 ± 15.7
	6	73.9 ± 20.6
	10	59.1 ± 15.7
Form 3	5	83.1 ± 11.1
	2	70.2 ± 13.9
	1†	60.0 ± 2.8

CSSAE = Clinical Simulation Self-Assessment Examination
*Values are mean ± SD
†Program 1 used the Secure Form with some, and Form 3 with others

Table 1 shows the demographic data reported by the students. The number of students enrolled in the programs ranged from 5 to 22, with a mean enrollment of 14. No significant differences were found in the distribution of gender, years of college experience, or months of clinical experience of the students in different programs. Using one-way analysis of variance (ANOVA or F test), there was a significant difference in the mean age of the students in different programs ($F = 2.37$, $p < 0.05$), where F = variance ratio. Post-hoc analysis revealed that the mean age of the students in Programs 1, 3, and 5 was higher than the mean age of the students in Program 9. Significant differences were also found in GPA between the students in different programs ($F = 2.61$, $p < 0.01$). The mean GPA of the students in Program 1 was higher than the mean GPA of the students in Programs 5, 6, and 10 ($p < 0.05$).

Table 2 shows the WGCTA scores. No significant differences were found in the CT scores of students in different programs ($F = 1.82$, $p > 0.05$).

Program directors selected the CSSAE form their students completed. Eight programs used the CSSAE Secure Form and 3 programs used CSSAE Form 3. Table 3 shows the DM scores from the CSSAE. The 110 students (from 8 programs) who completed the CSSAE Secure Form had a mean DM score of 85.9, with a range of 25–132. One-way ANOVA revealed significant differences in the DM scores of the students in different programs ($F = 5.57$, $p < 0.01$). Post-hoc analysis revealed that students in Program 10 had significantly lower DM scores than students in Programs 4, 8, 1, and 3. Because the probability of making a Type II error is influenced by sample size and the variability of the raw scores,[22] the absence of a significant difference in DM scores between students in Programs 7 and 10 may be due to the small number of students in those programs ($n = 9$ and $n = 7$, respectively) and the large variability and standard deviation of the scores of the students in Program 7. Students in Program 6 had significantly lower DM scores than students in Program 8. There was no significant difference between the DM scores of students in Programs 4 and 6, but that absence of significant difference may be due to the small number of students in Program 4 ($n = 5$).

Students who completed CSSAE Form 3 ($n = 33$) had a mean DM score of 77.8, with a range of 46–98. One-way ANOVA revealed a significant difference between the mean DM scores of the students in Programs 1, 2, and 5 ($F = 6.38$, $p < 0.01$). Post-hoc analysis revealed that students in Program 5 had significantly higher DM scores than students in Programs 1 and 2. There was no significant difference in DM scores between students in Programs 1 and 2.

Data Analysis

For the 110 students who completed the CSSAE Secure Form, a statistically significant correlation was found between CT and DM scores ($r = 0.32$, $p < 0.01$). For the 33 students who completed CSSAE Form 3, no significant relationship was found between CT and DM ($r = 0.21$, $p > 0.05$).

Statistically significant relationships were identified between age and CT ($r = 0.18$, $p < 0.05$), GPA and CT ($r = 0.40$, $p < 0.01$), and GPA and DM ($r = 0.325$, $p < 0.01$).

All participating program directors completed and returned the questionnaire indicating which strategies they used to develop CT and DM in their students (Table 4). The number of strategies used by programs ranged from 3 to 9. All the programs use case studies and clinical simulations, and over half of the programs also use role modeling, computer-assisted instruction, clinical patient rounds, small group discussion, and a study skills course to de-

Table 4. Strategies Used by Programs to Develop Critical Thinking and Decision-Making

Strategy	Critical Thinking (% of Programs)	Decision-Making (% of Programs)
Case studies	100	90
Clinical simulations	100	90
Role modeling by teacher	80	90
Computer-assisted instruction	80	80
Clinical patient rounds	70	60
Small group discussion	70	60
Study skills course	50	30
Problem-based learning	40	40
Other*	20	20

*One program reported that laboratory demonstration is used to develop both critical thinking and decision-making. One program indicated that a problem-solving model is used to develop both critical thinking and decision-making.

velop CT and DM. Four programs used problem-based learning, one program used laboratory demonstration, and another program used a problem-solving model. Table 5 shows the strategies used by each program. There were no apparent differences in the way the programs use the various strategies for developing CT and DM.

Discussion

The problem addressed in the present study is the challenge faced by respiratory care educators of improving students' DM skills in order to prepare practitioners for the advanced roles they will play in the future. Development of CT has been proposed as a method of improving DM, based on the hypothesis that there is a positive relationship between DM and CT.

The results of the present study may be helpful to educators in several ways. Because there is a positive relationship between DM and CT, programs may be able to improve students' DM skills by improving CT proficiency. Respiratory care programs could use information about CT as one of the criteria for selecting students and faculty, because individuals with strong CT proficiency may possess or develop better DM skills. But programs should be careful not to rely extensively on CT as an admission requirement. In the present study 90% of the participating programs reported the use of role modeling by the teacher as a strategy for developing CT and DM. This finding supports the need for teachers with strong CT skills. Although this study found a positive relationship between CT and DM in some students and in some programs, there was not always a significant relationship, and the correlational study design does not allow the inference of a causal relationship. Also, information about the strategies used to develop CT and DM can be valuable to educators as they seek new ways to improve the effectiveness of their programs and to provide students with the skills they need to be successful practitioners.

A statistically significant correlation was found between CT and DM for the 110 students who completed the CSSAE Secure Form ($r = 0.32$, $p < 0.01$). Cohen and Cohen would interpret the strength of that relationship as "medium" for research conducted in the behavioral sciences.[23] This correlation is similar to the correlations described in previous research. In studies of respiratory care students, Shelledy et al[6] reported a significant correlation of $r = 0.49$ and Mishoe et al[5] reported a significant correlation of $r = 0.34$. In a study of nursing students, Brooks and Shepherd[24] also reported a significant correlation ($r = 0.25$) between CT and DM.

Table 5. Program Use of Strategies to Develop Critical Thinking and Decision-Making

Strategy	Program									
	1	2	3	4	5	6	7	8	9	10
Case studies	X	X	X	X	CT	X	X	X	X	X
Clinical simulations	CT	X	X	X	X	X	X	X	X	X
Role modeling by teacher	X	X	X	DM	X	X	X	X	X	–
Computer-assisted instruction	–	X	–	X	X	X	X	X	X	X
Clinical patient rounds	–	X	–	X	X	X	X	X	–	CT
Small group discussion	–	X	–	X	X	X	X	X	CT	–
Study skills course	–	X	CT	X	CT	X	–	–	–	–
Problem-based learning	–	X	X	–	X	X	–	–	–	–
Other	–	X*	X†	–	–	–	–	–	–	–

X = critical thinking and decision-making
CT = critical thinking only
DM = decision-making only
*Laboratory demonstration
†Problem-solving model

When considering results from a correlational research design it is important to remember that although a statistically significant correlation allows us to predict the score of one variable based on the score of another variable, it does not allow us to infer a causal relationship between the variables.[19] Therefore, the results of the present study do not allow inference of a cause-and-effect relationship between CT and DM.

The programs surveyed incorporated between 3 and 9 strategies to develop CT and DM in their students. All programs used case studies and clinical simulations, and most programs also used role modeling, computer-assisted instruction, clinical patient rounds, small group discussion, and a study skills course. No differences were found in the way in which the programs use the different strategies for developing CT and DM.

All of the strategies included in the questionnaire were purported to improve CT and/or DM. No studies were found that compared the effectiveness of different strategies, and the present study did not identify any clear themes or patterns that suggest the superiority of particular strategies.

Program directors used the same strategies to develop both CT and DM, so it was not possible to examine the effect of the strategies individually on CT and DM. It is likely that CT and DM share some commonalties and may develop concurrently, as supported by the positive relationship observed in this study, but, again, a cause-and-effect relationship could only be established through additional study.

Although a significant relationship was found between age and CT, the correlation was small. The significant relationship between GPA and CT is expected because the WGCTA has been shown to correlate closely with other standardized tests of intelligence.[20] Mishoe et al also reported a significant correlation between GPA and CT,[5] which is expected because the CSSAE requires a strong background in discipline-specific content and therapeutic processes, which would generally result in good academic performance.

Conclusions

The findings support the belief that students with strong CT proficiency make better clinical decisions. Assessment of CT proficiency could be used as one part of a process for evaluating prospective students and faculty for respiratory care programs. Program directors and faculty felt that case studies and clinical simulations are important strategies. Further research should be conducted to increase the literature available to guide educators in developing CT and DM in respiratory care students. Although the present study involved 143 respiratory care students from 10 programs in 3 states, the number of students in each program was relatively small (mean of 14 students) and programs and subjects were not selected randomly. A larger study that incorporates random selection and more students would be useful. Additional suggestions for further study include use of an experimental design to identify a causal relationship, study of the effectiveness of the individual education strategies, a longitudinal study to examine changes in CT and DM over the course of study, and incorporating students from other allied health disciplines.

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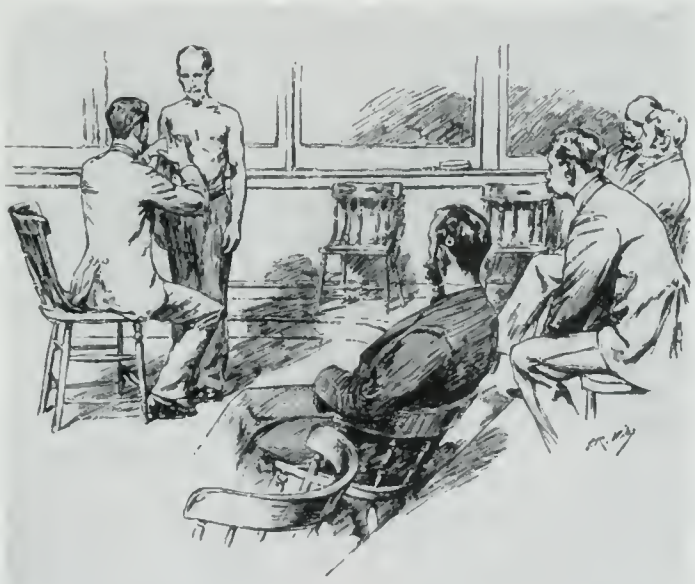
REFERENCES

1. Mishoe SC. Critical thinking, educational preparation, and development of respiratory care practitioners. Distinguished Papers Monograph 1993;2:29–43.
2. Cullen DL, Sullivan JM, Bartel RE, Fuson P, Galvin WF, Rinker RM, et al. Year 2001: delineating the educational direction for the future respiratory care practitioner. Proceedings of a national consensus conference on respiratory care education. Dallas: American Association for Respiratory Care, 1992.
3. Ennis RH. Critical thinking and subject specificity: clarification and needed research. Educational Researcher 1989;18(3):4–10.
4. Brooks KL. relationships among professionalism, critical thinking, decision making, and self concept for senior nursing students in four types of nursing curricula (dissertation). Philadelphia: Temple Univ; 1990.
5. Mishoe SC, Dennison FH, Thomas-Goodfellow L. A comparison of respiratory therapy students' critical thinking abilities with performance on the clinical simulation exam (abstract). Respir Care 1997; 42(11):1078.
6. Shelledy DC, Valley MA, Murphy DE, Carpenter ME. Effects of content, process, computer-assisted instruction, and critical thinking ability on students' performance on written clinical simulations. Respir Care Education Annual 1997;6:11–29.
7. Meyers C. Teaching students to think critically. San Francisco: Jossey-Bass; 1986.
8. Smith DG. College classroom interactions and critical thinking. J Educ Psychol 1977;69:180–190.
9. King PM, Kitchener KS. Developing reflective judgment: understanding and promoting intellectual growth and critical thinking in adolescents and adults. San Francisco: Jossey-Bass; 1994.
10. American Philosophical Association. Critical thinking: a statement of expert consensus for purposes of educational assessment and instruction: recommendations prepared for the committee on pre-college philosophy. ERIC Document Reproduction Service No. ED 315 423; 1990.
11. Brookfield S. On impostership, cultural suicide, and other dangers: how nurses learn critical thinking. J Contin Educ Nurs 1993;24(5):197–205.
12. Case B. Walking around the elephant: a critical thinking strategy for decision making. J Contin Educ Nurs 1994;25:101–109.
13. Kataoka-Yahiro M, Saylor C. A critical thinking model for nursing judgment. J Nurs Educ 1994;33(8):351–356.
14. Guning SC. Relationships among field independence, critical thinking ability, and clinical problem solving ability of baccalaureate nursing students (dissertation). Austin: Univ of Texas at Austin; 1981.

15. Holzemer WL, McLaughlin FE. Concurrent validity of clinical situations. West J Nurs Res 1988;10(1):73–83.
16. Kintgen-Andrews J. Critical thinking and nursing education: perplexities and insights. J Nurs Educ 1991;30(4):152–157.
17. Tanner CA. Instruction in the diagnostic process: an experimental study. In: Kim MJ, Moritz D, editors. Classification of nursing diagnoses: proceedings of the third and fourth national conferences. New York: McGraw-Hill; 1977:145–152.
18. LeCompte MD, Preissle J. Ethnography and qualitative design in educational research, 2nd ed. San Diego: Academic Press; 1993.
19. Krathwohl DR. Methods of educational and social science research: an integrated approach. New York: Longman; 1993.
20. Berger A. Review of the Watson-Glaser Critical Thinking Appraisal. In: Mitchell JV, editor. The ninth mental measurements yearbook. Lincoln NE: The Univ of Nebraska - Lincoln; 1985:1691–1693.
21. Clinical Simulation Self-Assessment Examination. Lenexa, KS: National Board for Respiratory Care and Applied Measurement Professionals; 2000.
22. Heiman GW. Basic statistics for the behavioral sciences. Boston: Houghton Mifflin; 1992.
23. Cohen J, Cohen P. Applied multiple regression / correlation analysis for the behavioral sciences, 2nd ed. Hillsdale NJ: Lawrence Erlbaum Associates; 1983.
24. Brooks KL, Shepherd JM. The relationship between clinical decision-making skills in nursing and general critical thinking abilities of senior nursing students in four types of nursing programs. J Nurs Educ 1990;29(9):391–399.



MAKING A DIAGNOSIS IN A CASE OF DISEASE OF THE LUNGS.

Courtesy of the National Library of Medicine

Bedside Spirometry in a Tertiary Care Hospital: The Cleveland Clinic Experience

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BACKGROUND: In contrast to outpatient, laboratory testing, which is performed by a small, dedicated group of pulmonary function technologists, inpatient, bedside spirometry at the Cleveland Clinic Hospital is performed less frequently and by a larger group of respiratory therapists with broader responsibilities. A 1998 audit of bedside spirometry tests at our hospital showed that American Thoracic Society acceptability and reproducibility criteria were infrequently met (15% of instances). METHODS: To address that shortcoming, we initiated an improvement plan for bedside spirometry that included: (1) A didactic review of American Thoracic Society acceptability and reproducibility criteria that was videotaped and reviewed by all but one of the therapists performing spirometry; (2) limiting the number of operators to a "core group" to allow more tests per therapist; (3) producing printouts of the pulmonary function tests, which allows immediate review of volume-time and flow-volume curves; (4) central review of all tests by a pulmonary function technologist and feedback and constructive suggestions on test quality and reproducibility to operators. After initiating the program we performed a consecutive survey of all inpatient spirometry sessions performed from July 16, 1998, to March 2, 1999. RESULTS: During the survey period, 63.5% of the tests (n = 137) were deemed acceptable, exceeding the low baseline rate of 15% ($p < 0.001$). Values for forced expiratory volume in the first second were reproducible in 83.9% of sessions. Values for forced vital capacity were reproducible in 80.3% of sessions. CONCLUSION: A quality improvement program for bedside spirometry testing that emphasizes training and routine feedback on test quality can enhance the quality of inpatient spirometry testing. *Key words: bedside spirometry, quality, pulmonary function testing, acceptability, inpatient.* [Respir Care 2002;47(5):578–582]

Introduction

Although spirometry is an important test that is performed frequently for outpatient assessment in both general and specialty practice[1] and in research,[2,3] inpatient applications are more limited. Nonetheless, experience at our 1,000-bed tertiary care center suggests that bedside spirometry is performed frequently in hospitalized patients and that, depending on the indication, the results may impact the patient's short-term management. As with all clinical tests, emphasis must be placed on assuring high quality testing so as to avoid interpretive and management errors resulting from technically flawed results.

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In the context of an ongoing interest in enhancing spirometry performance,[3,4] we sought to evaluate the frequency, indications for, and performance quality of bedside spirometry in our hospital. Specifically, as part of a program to assure high-quality bedside spirometry, we undertook a prospective evaluation of consecutive bedside spirograms in order to address several questions:

1. What is the total frequency of inpatient spirometry and what percentage of all inpatient tests require bedside performance?

2. What is the overall quality of bedside spirograms, based on American Thoracic Society standards[5] for spirometry testing?

3. What is the impact of a program to examine and enhance the quality of bedside spirometry?

4. Is there evidence of a threshold number of tests performed per technician to assure high quality?

Methods

All bedside spirograms at the Cleveland Clinic Hospital are performed by respiratory therapists, 86% of whom are registered respiratory therapists.

Spurred by the results of an audit of 20 bedside spirograms in early 1998, we implemented a program in July 1998 to enhance the quality of bedside spirometry. Key elements of the quality improvement program were as follows:

1. A policy was implemented that all inpatient spirometry should be performed in the pulmonary function laboratory whenever possible. Thus, bedside spirograms would be restricted to circumstances in which laboratory testing was not possible, such as when the patient was in an intensive care unit, was receiving infusions that precluded laboratory testing, or because the patient was admitted on a weekend or evening and was deemed to need urgent preoperative spirometry assessment sooner than laboratory testing was possible.

2. All bedside spirograms were centrally reviewed and graded for quality regarding acceptability and reproducibility criteria, and feedback was provided to the testing therapist.

3. Tutorial sessions conducted by pulmonary function laboratory technicians were offered to all respiratory therapists. Each session lasted 1 hour, was videotaped, and addressed how to perform the test, quality criteria (eg, start-of-test and end-of-test criteria), and details of the quality improvement program, including the need to print the spirograms and the plan to provide feedback on the printout. All but one of the respiratory therapists who performed inpatient spirometry testing either attended the session or reviewed the videotape. That individual was an experienced pulmonary function technician.

Bedside spirograms were performed using a Renaissance PB100 spirometer (Nellcor & Puritan Bennett/Tyco Healthcare, Pleasanton, California). All spirograms were printed on an attached portable printer as the spirometry maneuvers were performed, to allow the testing therapist to immediately evaluate the adequacy of each test. The therapists were encouraged to annotate the printouts regarding any problems or technical concerns with each bedside spirometry session. All the spirogram printouts were later submitted for central review to the technical director of the pulmonary function laboratory (KM), who has extensive experience in rating spirogram quality.[3]

Spirograms were rated according to the 1994 American Thoracic Society spirometry standards criteria,[5] including:

Table 1. Quality Rating System for Bedside Spirograms

- Each spirometry session consists of the 3 best spirograms (from a maximum of 8 tries) produced by the patient.
- Each of the 3 spirograms is rated on 3 features and given 1 point for each feature satisfied (for a maximum of 3 points per spirogram). These features are:
  - Extrapolated volume ≤ 5% of FVC or ≤ 0.15 L
  - No cough in first second
  - End-of-test criteria met[5]
- Thus, if each of the 3 spirograms satisfies all 3 features, 9 points are assigned for the session of 3 spirograms.
- Next, the 2 highest FEV_1 and FVC values from the 3 spirograms are compared.
  - If the 2 highest FEV_1 values are reproducible (ie, within 200 mL), 1 point is assigned to the session.
  - If the 2 highest FVC values are reproducible (ie, within 200 mL), 1 point is assigned to the session.
  - For example, if a session has 3 spirograms that satisfy all start-of-test and end-of-test criteria (9 points achieved) and also features the 2 largest FEV_1 and FVC values that are reproducible (ie, within 200 mL), then 11 points are assigned to the session.
- Finally, 1 point is assigned to the session if the therapist who performed the spirogram also annotated the hard copy of the spirogram with comments regarding the quality of the test (eg, patient's effort or ability to cooperate).
- Thus, the maximum number of points assigned for a spirometry session is 12 points.

FVC = forced vital capacity
FEV_1 = forced expiratory volume in the first second

1. a rapid "blast" at the beginning of exhalation: extrapolated volume/forced vital capacity ≤ 5% or extrapolated volume ≤ 0.15 L, whichever is larger;

2. no cough in the first second after a forced exhalation;

3. satisfactory end-of-test: a 1-second volume plateau after a smooth curvilinear rise or, in the absence of an expiratory plateau, an expiratory time of 15 seconds; and

4. reproducibility: serial measures of (A) forced expiratory volume in the first second (FEV_1) within 200 mL and (B) forced vital capacity (FVC) within 200 mL.

Based on tutorial sessions that reviewed the characteristics of an optimal bedside FVC maneuver and rating criteria, respiratory therapists were instructed to perform up to 8 maneuvers in each session, to obtain 2 that achieved the highest quality ratings. Post-bronchodilator testing was not performed at the bedside.

To provide an overall rating of the quality of bedside spirometry, we developed a spirometry rating score in which each spirometry session (consisting of the 3 best maneuvers obtained) was rated with an ordinal score (0–12 points). Table 1 shows the criteria for the rating system. To rate the session, 1 point was awarded to each effort satisfying each criterion: extrapolated volume within acceptable limits, freedom from cough in the first second of a forced exha-

Table 2. Reasons Spirometry Was Performed at the Bedside

Reason	Number	%
Late admission (after pulmonary function lab closed)	67	48.8
Reason not noted	33	24.1
Deemed unsafe to transport patient because patient was receiving medications that required close monitoring	14	10.2
Patient in intensive care unit	9	6.6
Physician ordered spirometry urgently (after 5 PM)	6	4.4
Physician ordered bed rest	4	2.9
Chest tube(s) in place	2	1.5
Patient to be discharged after test (pulmonary function laboratory unable to schedule soon enough)	2	1.5
Total	137	100.0

lation, and satisfaction of end-of-test criteria. After each of the 3 single best efforts was scored (total $\leq$ 9 points), additional points were awarded for FVC (1 point) and FEV_1 (1 point) reproducibility within 200 mL. Finally, 1 additional point was awarded if the spirogram printout was annotated regarding any shortcomings of the testing session. The maximum number of points that could be awarded to the testing session was 12.

Results

The quality program was implemented in July 1998. Between July 16, 1998, and March 2, 1999, inpatient spirometry was performed with 557 patients (mean 74 patients/mo), of whom 137 (25.5%) had spirometry performed at the bedside (mean ± SD age 62.4 ± 11.6 yr, 37% female). Those 557 patients represented a minority (6.6%) of all the patients (ie, both inpatients and outpatients) who underwent spirometry during the study period.

The most common reason for bedside testing was that the spirometry was ordered either urgently (4.4%) or on the day of admission (48.8%) but after the pulmonary function laboratory had closed for the day (Table 2).

The 137 bedside spirometry sessions were performed by 11 therapists, 5 of whom performed $\geq$ 10 bedside spirograms (mean 23.6 spirograms/operator) and 7 of whom performed < 10 bedside spirograms (mean 2.7 spirograms/operator) in this series.

The rate of achieving acceptable spirograms was significantly higher after the quality program was begun (63.5% vs 15%, $p < 0.001$). During the program, FEV_1 values were reproducible in 83.9% of sessions, and FVC values were reproducible in 80.3% of sessions. Freedom from start-of-test and end-of-test criteria violations was achieved in 80.3% and 63.5% of sessions, respectively. The mean ± SD spirometry quality score for the 137 bedside spirograms was 8.4 ± 2.4 (median 8.0, range 0–12).

By comparison with bedside testing, the rate of achieving acceptable spirograms in the 420 inpatients who underwent testing in the pulmonary function laboratory during the study period was higher (76.0%). Similarly, among a randomly selected sample of 440 outpatients tested in the Cleveland Clinic Foundation pulmonary function laboratory during the study period, the rate of achieving acceptable spirograms was 82.5%. Spirometry quality was also assessed for the 5 inpatients (of 137 [3.7%]) who underwent both bedside and laboratory spirometry during the same hospitalization. In all 5 of those patients, the laboratory test was performed later than the bedside test (mean ± SD interval 4 ± 2.1 d), usually to obtain confirmatory results. Acceptability criteria were met in 4 of the 5 laboratory sessions and in 3 of the 5 bedside sessions. Comparison of laboratory and bedside FEV_1 values showed agreement within 0.20 L in 2 of 5 patients. Mean ± SD laboratory FEV_1 values exceeded bedside values by 0.44 ± 0.57 L.

To evaluate whether performing a larger number of bedside spirograms was associated with better technical performance, we assessed the relationship between the rate of achieving acceptable tests and various indicators of quality (ie, the overall quality scores, the percent of sessions satisfying acceptability criteria, and the percent of sessions in which FEV_1 and FVC were reproducible). As summarized in Table 3, comparison of those rates between the therapists who performed < 10 bedside spirometry sessions and those who performed $\geq$ 10 showed no differences. Specifically, mean quality scores (8.3 ± 1.8 vs 8.4 ± 2.5, $p = 0.47$), the rate of achieving acceptable spirograms (68.4% vs 62.7%, $p = 0.82$), the rate of freedom from start-of-test criteria violations (78.9% vs 80.5%, $p = 0.44$), and the rate of freedom from end-of-test criteria violations (68.4% vs 62.7%, $p = 0.82$) were all similar.

We also performed a multivariate analysis in which the outcome measures were regressed against the number of test sessions performed per therapist, and no significant relationship was found (data not shown here).

Discussion

In this study of bedside spirometry quality after implementing a quality improvement program, we made the following observations:

1. Inpatient spirometry is commonly ordered in our large tertiary care hospital (74 patients/mo in this series). Among patients for whom inpatient spirometry was ordered in this series, clinical circumstances required bedside testing in only 26%.

2. The rate of achieving high-quality bedside spirograms was enhanced by a program of instruction and feedback on quality ratings of submitted spirograms for the therapists

Table 3. Comparison of Test Quality by Number of Spirograms Performed per Operator

	All Tests in Study $n = 137$ (%)	Tests/Operator < 10 $n = 19$ (%)	Tests/Operator ≥ 10 $n = 18$ (%)	p	Statistical Test
No start-of-test problem (extrapolated volume $\leq 5\%$ of FVC or ≤ 0.15 L)	110 (80.3)	15 (78.9)	95 (80.5)	0.44	McNemar's
No end-of-test problem	87 (63.5)	13 (68.4)	74 (62.7)	0.82	chi-square
Reproducible FVC	110 (80.3)	17 (89.5)	93 (78.8)	0.439	chi-square
Reproducible FEV_1	115 (83.9)	17 (89.5)	98 (93.1)	0.711	chi-square
Both FVC and FEV_1 reproducible	104 (75.9)	16 (84.2)	88 (74.6)	0.301	chi-square
Mean session score ± SD	8.4 ± 2.4	8.3 ± 1.8	8.4 ± 2.5	0.474	M-W‖ rank sum
Acceptable sessions	87 (63.5)	13 (68.4)	74 (62.7)	0.824	chi-square

FVC = forced vital capacity
FEV_1 = forced expiratory volume in the first second
‖ M-W = Mann-Whitney

who performed the measurements. Specifically, in this "before-after" observational cohort study, the rate of achieving acceptable spirograms increased 4-fold (from 15% to 63.5%, $p < 0.001$) after implementing the program. In addition, high rates of reproducibility for FEV_1 and FVC measurements were achieved after implementing the quality improvement program.

3. Using an a priori threshold of 10 spirometry sessions per therapist, our series failed to show a higher rate of achieving acceptable and reproducible tests among therapists performing ≥ 10 tests than among those performing < 10. Thus, we were unable to show a training effect for performing bedside spirometry.

Although the present study is, to our knowledge, the first to assess inpatient spirometry, our findings both confirm and extend experience with outpatient office spirometry. Specifically, in examining the quality of spirometry performed in primary care practices, Eaton et al[1] reported that only 12.5% of spirogram sessions achieved ≥ 2 maneuvers meeting acceptability criteria. Performance was enhanced significantly after operators underwent a 2-hour training workshop. For example, the rate of achieving ≥ 2 acceptable maneuvers was higher in the trained than in untrained operators (33.1% vs 12.5%, $p < 0.0001$), as was the rate of achieving ≥ 2 reproducible maneuvers (13.5% vs 3.4%, $p < 0.0001$).

As with those observations regarding office spirometry in primary care practice, our study shows similarly low rates of achieving acceptable spirograms before implementing a training and quality improvement program (15%) and a significantly higher rate (63.5%) afterward. Thus, our findings suggest that, as with office spirometry, achieving high-quality bedside spirometry requires training, ongoing quality monitoring, and feedback to the operator.

Two general methods for providing feedback on spirometry quality have been described:

1. Point-of-care feedback by "smart" spirometers that have built-in prompts regarding acceptability and reproducibility, and

2. Central review of submitted spirograms, with subsequent feedback to the operators.

Though comparison of those 2 feedback approaches has not, to our knowledge, been undertaken, both strategies have been effective. For example, feedback from "smart" spirometers was provided in the study of office spirometry by Eaton et al,[1] in which training and monitoring effected a nearly 3-fold increase in achieving 2 acceptable expiratory efforts. In the research setting, the Lung Health Study[2] achieved a 97.9% rate of sessions with acceptable and reproducible spirograms, using both point-of-testing feedback and central review. Similar reproducibility rates were achieved using central review only in the Registry of Patients with α_1-Antitrypsin Deficiency.[3] Though assessing the elements of an optimal quality monitoring program was beyond the scope of the present study, our findings certainly suggest that an ongoing review process is important.

Several aspects of the present study warrant further comment. First, the baseline rate of achieving acceptable spirograms was low. Indeed, that low initial rate was the impetus for the quality improvement program and for this observational study. Many reasons for the initial poor quality of inpatient spirometry can be imagined, especially because that low rate of achieving acceptable spirograms differs from the high rate in outpatient laboratory testing (82.5% in this series). One likely reason for that difference is that outpatient testing is performed in a pulmonary function laboratory, which features a small, dedicated staff who undergo ongoing training and quality monitoring. In contrast, before this study was undertaken, responsibility for inpatient spirometry testing in our hospital was shared among a large group of therapists who were trained to perform spirometry as part as their initial instruction but

who had no subsequent formal training or quality monitoring.

A second factor that might explain the low pre-study acceptability rate is that neither printouts nor graphic displays of spirograms were initially available, precluding visual inspection of the spirogram by the operator. Because quality monitoring requires visual assessment, that lack of graphic feedback may have contributed to the low rate of achieving acceptable and reproducible spirograms.

A third reason for the difference in quality between outpatient laboratory and inpatient bedside spirometry may be the greater experience among outpatient pulmonary function technicians than among hospital-based respiratory therapists. Specifically, in our pulmonary function laboratory, which performed 7,814 spirometry tests during the study period, the mean number of spirograms performed per technician (1,302) greatly exceeded the mean of 12.4 spirograms per operator in the current inpatient series.

Given the premise that greater testing experience should translate to better quality testing, our observation that the quality ratings did not differ between therapists performing ≥ 10 and those performing < 10 is surprising. Several potential reasons for this failure to observe a training effect can be proposed. First, rather than exploring the data for a specific threshold number, we chose 10 spirometry sessions as an a priori threshold. Though our multivariate analysis did not show a training effect, it is still conceivable that a threshold value other than 10 may better identify a critical number of spirometry sessions above which quality improves. Second, it is possible that the training provided to all therapists as an early part of the quality improvement program (and which included hands-on testing) may have ablated any difference between the low-volume and higher-volume testers. In support of that explanation, we also observed no difference in quality ratings between the first half and the second half of the 137 spirometry sessions performed (58.8% vs 69.1%, $p = 0.284$).

A limitation of the present study is that we did not track the clinical decisions made based on bedside spirometry results, so we cannot assess how often suboptimal spirometry results prompted flawed clinical decisions. However, our inpatient experience clearly shows that underestimated FEV_1 values from bedside spirometry can have adverse consequences, such as delays of elective operations, additional consultation, and additional laboratory testing (eg, arterial blood gases and repeat laboratory spirometry when possible). Indeed, this experience has caused us to generally recommend laboratory spirometry when preoperative pulmonary function testing is indicated.

Conclusions

Our results suggest that, like office spirometry, the quality of bedside spirometry should be monitored and can be enhanced markedly by implementing a quality improvement and monitoring program. Important elements of such a quality program include formal training of those performing spirometry and ongoing feedback to the operators regarding spirometry quality, ideally including both central review of submitted spirograms and point-of-testing feedback.

REFERENCES

1. Eaton T, Withy S, Garrett JE, Mercer J, Whitlock RM, Rea HH. Spirometry in primary care practice: the importance of quality assurance and the impact of spirometry workshops. Chest 1999;116(2): 416–423.
2. Enright PL, Johnson LR, Connett JE, Voelker H, Buist AS. Spirometry in the Lung Health Study. 1. Methods and quality control. Am Rev Respir Dis 1991;143(6):1215–1223.
3. Stoller JK, Buist AS, Burrows B, Crystal RG, Fallat RJ, McCarthy K, et al. Quality control of spirometry testing in the registry for patients with severe α_1-antitrypsin deficiency. α_1-Antitrypsin Deficiency Registry Study Group. Chest 1997;111(4):899–909.
4. Stoller JK, Basheda S, Laskowski D, Goormastic M, McCarthy K. Trial of standard versus modified expiration to achieve end-of-test spirometry criteria. Am Rev Respir Dis 1993;148(2):275–280.
5. American Thoracic Society. Standardization of spirometry, 1994 update. Am J Respir Crit Care Med 1995;152(3):1107–1136.

A Survey of Physicians to Identify Their Expectations of Respiratory Therapists in Patient Assessment

Robert L Wilkins PhD RRT FAARC, Jacqueline Evans CRT, and Lennard Specht MD

INTRODUCTION: The exact role of respiratory therapists (RTs) in patient assessment is not well defined. The purpose of this study was to identify local physician expectations of RTs for the application and interpretation of various assessment procedures. METHODS: A survey was developed that called for physicians to indicate on a scale of 1 to 5 (1 = never, 2 = rarely, 3 = occasionally, 4 = often, and 5 = always) how important they perceive it is for RTs to be able to interpret or perform 20 commonly used patient assessment tests or procedures. The survey was mailed to 25 physicians who care for patients in the medical and surgical intensive care units at Loma Linda University Medical Center and the Jerry L Pettis Veterans Administration Memorial Medical Center. RESULTS: Twenty (80%) of the surveys were returned. The assessment procedures rated highest were: to assess lung sounds (4.8), to interpret arterial blood gas values (4.5), to interpret pulmonary function tests (4.4), to assess and interpret vital signs (4.4), and to interview patient to clarify symptoms (4.3). The procedures consistently rated low were: to interpret echocardiography (2.5), to assess abdomen (2.3), to interpret sleep studies (2.2), and to interpret electroencephalogram (1.7). A total expectation score for each completed survey was determined by adding the scores from each of the 20 assessment procedures. The average ± SD total score was 66.8 ± 10.4 on a scale of 20 to 100. The total expectation score did not vary by years of experience in patient care or type of medical specialty. CONCLUSIONS: The results indicate that the physicians completing our survey generally have high expectations of RTs for the application of patient assessment techniques that involve direct evaluation of lung performance or condition. *Key words: patient assessment, physician expectations, survey, respiratory therapist.* [Respir Care 2002;47(5):583–585]

Introduction

Respiratory care by protocol appears to be growing in popularity,[1–3] which calls for respiratory therapists (RTs) to be skilled in patient assessment. The exact role of RTs in patient assessment, however, is not well defined and probably varies by location around the country. The expectations of physicians may be a key factor in determining the role of RTs in the assessment process, since RTs work under the direction of physicians. Educators of respiratory care students need to know physician expectations in order to provide students with the knowledge and skills they will need to be effective in patient care.

The purpose of this study was to identify local physicians' expectations of RTs in patient assessment. We attempted to answer 3 questions:

1. What are local physicians' expectations of RTs regarding various patient assessment procedures and tests?
2. Are there differences in expectations based on medical specialty (eg, pulmonologists vs physicians of other medical specialties)?
3. Is there a relationship between a physician's years of experience and his or her expectations of RTs in patient assessment?

We hypothesized that pulmonologists would have different expectations than other physicians, because of their close relationship with RTs. In addition we expected that, because of their exposure to RTs in the clinical setting, physicians with many years of experience would have greater expectations than those with fewer years of experience.

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Table 1. Survey Items Listed by Physician Ranking

Assess lung sounds	4.8 ± 0.41
Interpret arterial blood gas values	4.5 ± 0.51
Interpret pulmonary function tests	4.4 ± 0.81
Assess vital signs	4.4 ± 0.93
Interview patient to clarify symptoms	4.3 ± 1.01
Percuss chest to assess resonance	4.1 ± 1.07
Assess tactile fremitus	4.0 ± 1.23
Interpret chest radiograph	3.5 ± 0.60
Interpret hemodynamics	3.4 ± 1.19
Interpret sputum Gram stain and culture	3.3 ± 1.30
Assess heart sounds	3.3 ± 1.10
Assess pedal edema	3.2 ± 1.00
Interpret complete blood count	3.2 ± 1.30
Interpret electrolytes	3.1 ± 0.99
Interpret electrocardiogram	2.8 ± 0.69
Interpret cardiac enzymes	2.6 ± 1.23
Interpret echocardiography	2.5 ± 0.88
Assess abdomen	2.3 ± 0.81
Interpret sleep studies	2.2 ± 1.00
Interpret electroencephalogram	1.7 ± 0.85

Values are rank ± SD

Methods

We developed an in-house survey with the assistance of the medical director of the respiratory care program at Loma Linda University, 2 experienced RTs, and a faculty member with more than 20 years of teaching experience in respiratory care. The survey called for physicians to rate 20 patient assessment procedures based on how important they believe it is for RTs to perform or interpret each procedure (Table 1). The items listed in the survey represented a broad spectrum of valid patient assessment procedures RTs may or may not traditionally perform in clinical practice. The frequency with which RTs perform these tests differs widely, from *commonly* to *only with specialized training*. The physicians were asked to rate each item on a scale of 1 to 5, in which 1 = never, 2 = rarely, 3 = occasionally, 4 = often, and 5 = always. Approval was obtained from the institutional review board at Loma Linda University prior to mailing the surveys.

The survey was mailed to the 25 physicians who regularly attend to critically ill patients in the medical and surgical intensive care units at Loma Linda University Medical Center and The Jerry L Pettis Veterans Administration Memorial Medical Center. The subjects chosen for this survey were the primary attending physicians who admit and supervise the care of patients in intensive care units where RTs are very active. The target participants included 11 pulmonary/critical care physicians and 14 other physicians (surgeons and anesthesiologists). The survey was completed anonymously by the physicians and returned to us for analysis.

A total expectation score was determined for each completed survey by adding the scores from each of the 20 assessment procedures. This total expectation score was used to identify if a correlation existed between the physician years of experience and overall expectations and to further identify if there was a difference in expectations based on medical specialty. Reliability analyses were done to determine the standardized item alpha for the 20 items measured in the survey.

Results

Twenty completed surveys (80%) were returned. Table 1 shows the average scores of the physicians' ratings for each of the 20 procedures listed in the survey. Assessment procedures with scores of ≥ 4.0 represent skills the participating physicians believe RTs should be able to apply "often" or "always" in the clinical setting. Items with scores of < 3.0 represent skills that our participants believe RTs do not need to apply to patient care on a frequent basis.

The total expectation score average ± SD was 66.8 ± 10.4. Reliability analyses revealed a standardized item alpha for the 20 items in the survey to be 0.89, indicating that summing the 20 items for a total expectation score was appropriate. No relationship was found between years of experience and total expectation score ($r = 0.108$, $p = 0.660$). Similarly, the total expectation score did not vary by medical specialty of the participating physicians.

Discussion

The majority of the assessment procedures rated highest included skills associated with direct evaluation of the lungs (eg, lung auscultation, interpretation of arterial blood gas values, interpretation of pulmonary function tests). Exceptions include interviewing patients and assessment of vital signs, which do not directly measure lung function but are often used to gain an understanding of the health status of pulmonary patients. Therefore it is important for educators to emphasize interviewing and assessment of vital signs in their curricula, in addition to those assessment procedures directly associated with evaluation of lung performance.

The assessment procedures consistently rated lowest in our survey are those involving evaluation of systems other than the respiratory system (eg, interpret electroencephalogram, interpret echocardiogram, assess abdomen). These results suggest that the physicians participating in our study do not hold RTs accountable for performing assessment procedures that evaluate body systems other than the respiratory system. As RTs become more involved with multiskilling, steps may be needed to increase physician expec-

tations. We speculate that this may indicate a need for further education of RTs in assessing other systems, if expectations are to increase in the future.

The average total expectation score of 66.8 indicates that, in general, the physicians in our survey have high expectations of RTs in patient assessment. That score would have been higher had we omitted procedures not associated with the routine practice of respiratory care (eg, echocardiography). The total expectation score did not vary by years of experience or medical specialty, which suggests that recent medical school graduates have expectations similar to their senior peers and that expectations are similar across disciplines. Our hypotheses that pulmonary physicians would have different expectations than physicians of other medical specialties and that years of experience would influence physicians' assessment expectations were not substantiated by our results.

The major limitation of this study is the sample size. Our results reflect the opinions of only 20 physicians in several local intensive care units and cannot be generalized to physicians in other regions across the country. A larger study should be done to gain a national perspective. Faculty of respiratory therapy schools may want to conduct similar surveys in their local areas to identify which assessment procedures RTs are expected to perform in their communities of interest, which would be useful for curriculum development.

Conclusions

The physicians participating in our survey have high expectations of RTs in patient assessment, especially in those techniques that involve direct evaluation of the patient's pulmonary status. Clearly the physicians we surveyed expect RTs to apply a variety of assessment techniques to patient care. This high expectation was present in pulmonologists and physicians of other medical specialties practicing in 2 Southern California medical centers and did not vary by years of experience. We speculate that RT education may need to be stronger in assessment techniques not directly associated with the lungs if future physician expectations of RTs are to increase in that domain.

REFERENCES

1. Pierson DJ. The future of respiratory care. Respir Care 2001;46(7): 705–718.
2. Stoller JK, Mascha EJ, Kester L, Haney D. Randomized controlled trial of physician-directed versus respiratory therapy consult service-directed respiratory care to adult non-ICU inpatients. Am J Respir Crit Care Med 1998;158(4):1068–1075.
3. Kollef MH, Shapiro SD, Clinkscale D, Cracchiolo L, Clayton D, Wilner R, Hossin L. The effect of respiratory therapist-initiated treatment protocols on patient outcomes and resource utilization. Chest 2000;117(2):467–475.

Chronic Obstructive Pulmonary Disease: On an Exponential Curve of Progress

John E Heffner MD

Introduction
Societal Impact of Chronic Obstructive Pulmonary Disease
Response to the Epidemic of Chronic Obstructive Pulmonary Disease
Definition
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Acute Exacerbations of Chronic Obstructive Pulmonary Disease
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Future Directions

The last 50 years have witnessed major progress in our understanding of the underlying nature of chronic obstructive pulmonary disease (COPD) and approaches to care. During the last 10 years, however, there has been an explosive expansion of knowledge related to this disorder. Large-scale epidemiologic studies show that COPD is the fourth leading cause of death in the United States and will be the fifth leading health burden worldwide by the year 2020. We have also learned that COPD is an inflammatory disorder of small airways, which presents new opportunities for therapy involving interfering with the early components of the inflammatory cascade. Unfortunately, most clinicians fail to perform spirometric screening of at-risk individuals, so most patients first present with COPD during a sudden exacerbation of the disease. Worldwide interest is emerging in promoting earlier diagnosis and finding new pharmacologic interventions to halt the progression of airway and parenchymal damage. In this review, evidence is presented that we are on the upward limb of an exponential curve of progress in managing COPD, which will produce major advances in our ability to recognize and treat this disorder in its earliest stages. *Key words: chronic obstructive pulmonary disease, COPD.* [Respir Care 2002;47(5):586–607]

Introduction

To know chronic obstructive pulmonary disease (COPD) is to know pulmonary—and a large chunk of critical care—medicine. Those of us who were trained in pulmonary critical care during the 1970s gained much of our respiratory education through caring for COPD patients. We learned ventilator management and the importance of intrinsic positive end-expiratory pressure, principles of re-

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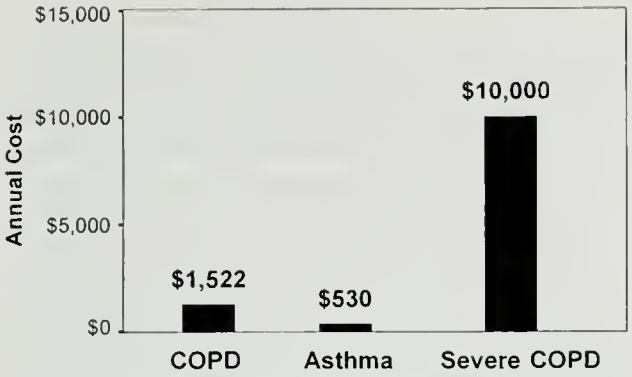

Fig. 1. Annual health expenditures for all chronic obstructive pulmonary disease (COPD) patients, asthma patients, and patients with severe COPD.

spiratory care, bronchodilator and corticosteroid pharmacotherapy, management of drug toxicity (thanks to the use of aminophylline), the importance of measuring health-related quality of life in monitoring disease, and the need for physicians to become skilled in palliative and end-of-life care.

So I appreciate the opportunity at this stage of my career to pause and reflect on the progress we have made in our understanding of COPD. As I look back at the 30 years of my experience with this condition I conclude that we are on an exponential curve of improving our knowledge of COPD. In this paper I will highlight that progress by touching on those aspects of our understanding of COPD that have undergone the greatest change and those that offer the most promise for providing further insight into the disease.

Societal Impact of Chronic Obstructive Pulmonary Disease

This country has traditionally under-recognized the societal impact of COPD. Emerging health care statistics are now characterizing the true proportions of the COPD epidemic. Sixteen million Americans have symptomatic COPD.[1] COPD now accounts for 4% of all deaths in the United States,[2] making it the fourth leading cause of death. Between 1966 and 1995, age-adjusted death rates for stroke and coronary heart disease declined by 58% and 45%, respectively, whereas the death rate from COPD increased by 71%,[3] making it the only leading cause of mortality that is increasing in incidence.[4] The annual cost of caring for a COPD patient in the United States is $1,522, which is almost 3 times the per capita cost of caring for an asthma patient (Fig. 1).[5] The annual cost of care for a patient with severe COPD is $10,000.[6] Expenditures for Medicare beneficiaries who have COPD are nearly 2.5 times higher than expenditures for those without ($8,482 vs $3,511).[7] COPD costs the United States $14.5 billion a year.[8]

The burden of COPD is even greater when examined from a global perspective. The World Health Organization estimates that COPD accounts worldwide for 1 million years of life lost per annum and 29 million disability-adjusted life-years.[9] By the year 2020 COPD will be fifth among the diseases that present a burden to society.[10]

Recent models indicate that the health care impact of COPD will only worsen in the future.[11] An aging population and past smoking behavior destine the prevalence of COPD to rise, with a greater impact on women than men.[12-14] Regardless of future changes in smoking patterns, the die is cast; we will see more COPD patients in coming years.[11,13,14]

Response to the Epidemic of Chronic Obstructive Pulmonary Disease

The world is beginning to respond to this global epidemic, as shown by the publication since 1995 of 13 COPD clinical practice guidelines by multiple professional societies (Table 1),[15] including the recently published National Heart Lung and Blood Institute/World Health Organization Global Initiative for Chronic Obstructive Lung Disease (GOLD).[16] These guidelines represent an important "coming together" of worldwide experts in developing a consensus approach to this condition. This consensus promises to increase public and policymaker awareness of the threat of COPD to global health, assist implementation of effective programs for its diagnosis and management, and promote programs to prevent its occurrence.

Definition

COPD is a general term that encompasses respiratory conditions—most commonly chronic bronchitis and em-

Table 1. Guidelines on Chronic Obstructive Pulmonary Disease Published 1995 through 2001

American Thoracic Society
British Thoracic Society
Canadian Thoracic Society
Die Deutsche Atemwegsliga
European Respiratory Society
Norwegian Institute of Pharmacotherapy
Polish Phthisiopneumonological Society
Société de Pneumologie de Langue Française
Spanish Society of Pneumonology and Thoracic Surgery
Swiss Society of Pneumology
Thoracic Society of Australia and New Zealand
United States National Heart, Lung, and Blood Institute/World Health Organization - Global Initiative for Chronic Obstructive Lung Disease (GOLD)
American College of Physicians and American Society of Internal Medicine

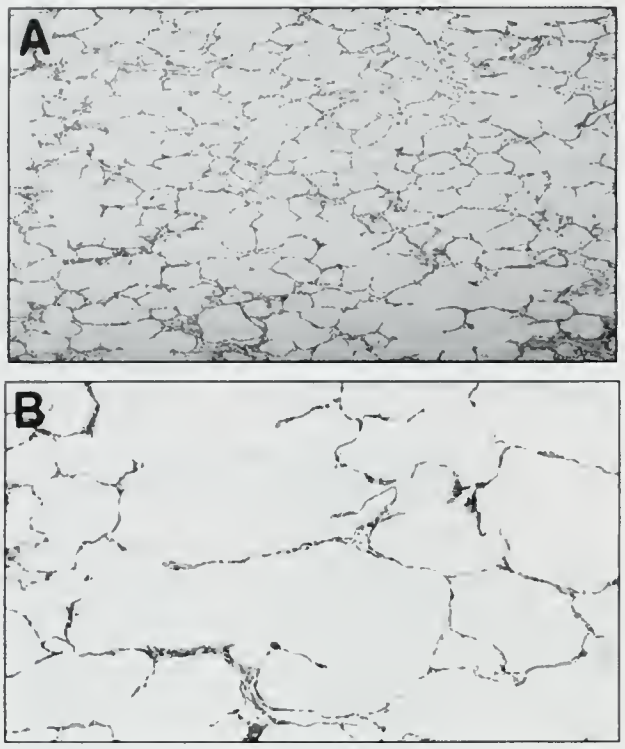

Fig. 2. Lung specimens showing (A) normal lung and (B) lung affected by severe chronic obstructive pulmonary disease (COPD). The COPD lung shows a loss of alveolar tissue and supporting structures. No evidence of fibrosis is present in this section.

physema—that damage small and large airways, destroy lung parenchyma, and produce expiratory air flow limitation, which is the pathophysiologic hallmark of the disease. Chronic bronchitis begins in the small airways as an inflammatory condition that spreads to larger airways, causing mucus hypersecretion, increased risk of bacterial bronchial infections, and air flow limitation. Emphysema is defined by the presence of irreversible lung damage from the dissolution of alveolar tissue in the absence of marked fibrosis and from reduced lung elasticity that causes expiratory airway collapse and air flow limitation (Fig. 2).

During recent years the definition of COPD has evolved in several ways. Asthma, because of its unique pathophysiology, is no longer included under the definition. Classic definitions of COPD have rested on physiologic measures of air flow limitation. The practice guideline developed by the European Respiratory Society (ERS) describes COPD as a "disorder characterized by reduced maximum expiratory flow and slow forced emptying of the lungs."[17] The American Thoracic Society (ATS) statement on COPD defines it as a condition characterized by the progressive development of air flow limitation that is not fully reversible.[18] Recent advances have identified COPD as an inflammatory condition caused by the inhalation of proinflammatory particulates. In response to this enhanced pathophysiologic understanding, the GOLD initiative expanded the definition of COPD to "a disease characterized by a progressive air flow limitation caused by an abnormal inflammatory reaction to the chronic inhalation of particles."[16] Changes in the definition of major disorders are hard earned and reflect major progress in our understanding of underlying disease processes.

The general definition of COPD, however, remains difficult to apply to individual patients, a fact that explains the common misdiagnosis of adults with chronic respiratory conditions. Patients with cough and dyspnea who smoke are often misdiagnosed as having COPD when they actually have asthma, and nonsmoking patients who develop COPD from noncigarette-related causes are frequently misdiagnosed as asthmatics.[19] Some patients seen in clinical practice present with air flow limitation that has characteristics of both asthma and COPD and defies a specific diagnosis.[20,21] Attempts to differentiate between asthma and COPD by the degree of air flow limitation, reversibility of airway obstruction, or nature of the airway inflammatory reaction prove unreliable in many older patients with respiratory complaints.[19]

The absence of consensus regarding the degree of expiratory flow limitation required to establish the presence of "air flow limitation" further complicates the diagnosis of asthma in individual patients. The 1995 ATS statement on COPD does not define a numerical value that establishes the presence of obstructive lung disease,[18] although an earlier ATS statement on lung impairment and disability proposes an FEV_1/FVC (ratio of forced expiratory volume in the first second to forced vital capacity) $< 75\%$.[22] The ERS definition of airway obstruction requires an FEV_1/VC (ratio of forced expiratory volume in the first second to slow vital capacity) $< 88\%$ of the predicted value for men and $< 89\%$ of predicted for women.[17] Most clinical trials of drugs for COPD use an $FEV_1/FVC < 70\%$ to define airway obstruction. The Canadian Thoracic Society guidelines[23] do not provide functional definitions of air flow limitation. The Thoracic Society of Australia and New Zealand reported different values for FEV_1/VC ($< 70\%$) and FEV_1/FVC ($< 75\%$).[24] The British Thoracic Society[25] offered presence of both a reduced FEV_1 ($< 80\%$ of predicted) and an $FEV_1/VC < 70\%$.

The difference in the definitions of COPD has a profound effect on estimates of the prevalence of the disease in population-based studies. Viegi et al recently applied the ERS and ATS definitions of air flow limitation to a population in northern Italy.[26] The prevalence of COPD ranged from 11.0% using the ERS definition to 40.4% using the ATS criterion. The publication of the GOLD initiative, however, promises to standardize the definition of air flow limitation, thereby promoting more precise estimates of disease prevalence.

Epidemiology

In the last several years we have advanced our understanding of the prevalence of COPD and its impact on public health. Early prevalence data derived from studies of hospitalized patients with severe or end-stage disease.[27] Epidemiologic studies from the 1960s through the early 1980s were regional and used different definitions of COPD. Consequently, estimates of COPD prevalence encompassed a broad range, from 3% to over 20% for men and from 6% to 14% for women.[28–30] Incomplete analyses of confounding conditions, absence of age adjustments, use of administrative databases with incomplete information capture, and revisions to the International Classification of Disease codes, which alter disease definitions, further limited those early studies. Those limitations resulted in underestimation of the prevalence and impact of what now appears to be an under-diagnosed condition. Only 3.5% of randomly selected patient records from the 1992 Medicaid database provided sufficient information to establish a diagnosis of COPD, despite contemporary population-based studies suggesting that the prevalence is at least 10%.[7] Considering the impact of COPD on global health, the National Heart, Lung, and Blood Institute stated, "It is . . . essential that strategies are implemented on a global scale to assess the prevalence of COPD. . . ."[31]

We are now seeing more advanced strategies to estimate the prevalence of COPD. The third National Health and Nutrition Examination Survey (NHANES III) is a population-based study of 16,084 smokers and nonsmokers. The study defined obstructive lung disease as $FEV_1/FVC < 70\%$.[32] It examined the study population for the prevalence of chronic bronchitis, asthma, and emphysema, and found an overall prevalence that ranged from 10% to 18%, with a higher prevalence in whites than in blacks.

The NHANES III study showed that air flow obstruction—regardless of smoking history—is common in the general population of the United States and that air flow limitation is associated with impaired health and functional status.[33] Also, undiagnosed air flow obstruction was more common than doctor-diagnosed COPD and asthma combined.[33] After excluding patients who carried a physician diagnosis of air flow limitation, 12% of the general population had measurable air flow obstruction.

Most of the undiagnosed cases of air flow limitation in the NHANES III study were found in younger patients who had mild disease.[33] That observation supports the belief that health interventions, such as health care screening, in younger patients offer the greatest potential benefit for preventing obstructive lung disease.

The IBERPOC (Estudio Epidemiolólico de la EPOC en España) Project provides us with greater precision in estimating the incidence of air flow limitation in a general population relative to smoking history.[34] The IBERPOC population-based study included 4,035 persons 40–69 years of age, identified randomly. The prevalence of COPD was 9.1%, with a prevalence of 26% in current smokers and 24% in ex-smokers. Similar to the NHANES III study, only 22% of patients found to have air flow limitation carried diagnoses of COPD. That under-diagnosis of the disease occurred despite the presence of respiratory symptoms in 48% of patients who had air flow limitation.

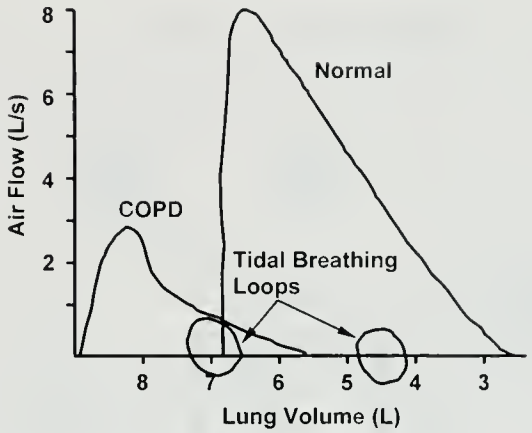

Fig. 3. Flow-volume loops and curves from a patient with severe chronic obstructive pulmonary disease (COPD) and from a normal person. The COPD patient approaches maximum air flow during tidal breathing.

It has traditionally been stated that only 10–15% of smokers develop COPD.[35,36] The results of the NHANES III study and the IBERPOC Project, however, suggest that the observed occurrence rate would be higher were data analyzed across older age ranges. A recent study using a model based on smoking rates found that the prevalence of COPD increased from 10% to 43% among current smokers as the age range increased.[37] A study in Poland, which used spirometric screening of at-risk patients who were > 39 years old with a smoking history > 10 years, found evidence of air flow limitation in 31%.[38]

These sobering statistics support the contention that COPD is under-diagnosed and that we need better classification methods to identify persons at risk for COPD and to develop and disseminate public health strategies to reduce the burden of this disease.

Pathogenesis

Dyspnea represents the most fundamentally important and disabling feature of COPD and occurs as a consequence of air flow limitation. The maximum pulmonary ventilation that a person can achieve is limited by the maximum flow rate that can be generated.[39] Healthy individuals do not reach their limits of expiratory air flow even with strenuous exercise. In contrast, as shown by Figure 3, patients with severe COPD may exhibit air flow limitation

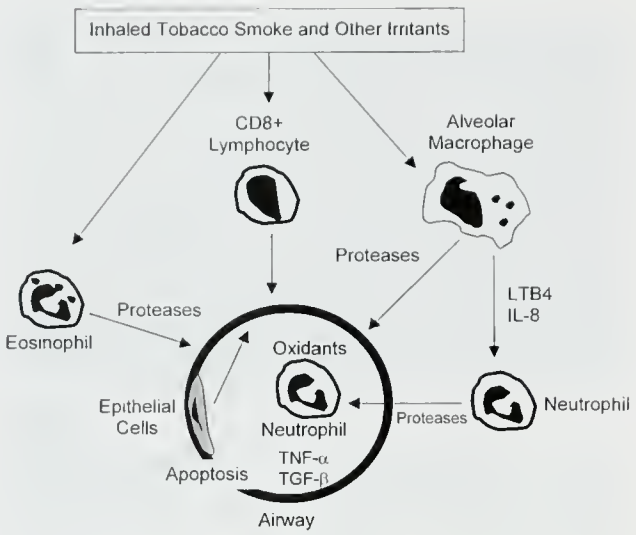

Fig. 4. Evidence for the inflammatory nature of chronic obstructive pulmonary disease. Inhaled proinflammatory smoke and other irritants activate alveolar macrophages, CD8+ lymphocytes, and possibly eosinophils, epithelial cells, and alveolar cells. Activated cells release proteases and neutrophil chemotactic factors (leukotriene B4 [LTB4] and interleukin 8 [IL-8]) that recruit phagocytes to the airway. Epithelial cells are subject to apoptosis as a direct result of inhaled toxins. Tumor necrosis factor alpha (TNF-α) and transforming growth factor beta (TGF-β) concentrations increase in the airways.

even at resting tidal ventilation.[40] These patients have no ventilatory reserve and breathe along their maximal expiratory flow curve. With increased workloads, patients with severe air flow limitation experience dynamic hyperinflation, with an attendant increase in inspiratory work.[40,41] Dynamic hyperinflation impairs inspiratory muscle function, produces adverse effects on hemodynamics, and worsens dyspnea.[42,43]

Although air flow limitation is well established as the primary cause of COPD symptoms, the structural source of air flow obstruction remains incompletely defined. We know that abnormalities in airway structure, such as goblet cell metaplasia, inflammation, smooth-muscle hypertrophy, and fibrosis, narrow airway caliber and reduce air flow. Destruction of alveolar walls diminishes the attachments of alveoli and reduces lung elastic recoil, which decreases expiratory driving pressure and produces small airways collapse during forced exhalation.[44] But the degree of air flow limitation observed in COPD is not entirely explained by those abnormalities. Many experts suspect that other factors, such as peribronchiolar inflammation or fibrosis of small airways, contribute to air flow limitation.[45]

A wealth of data supports the concept that ongoing airway and parenchymal inflammation in response to inhaled particulates is the primary cause of lung destruction in COPD (Fig. 4).[46] Inflammatory cytokines such as interleukin 8 (IL-8), leukotriene B4, other neutrophil chemotaxins, and tumor necrosis factor α (TNF-α) are higher in induced sputum from COPD patients.[47,48] There are also more inflammatory cells in sputum from COPD patients, and the number of inflammatory cells correlates with the severity of air flow limitation.[48–50] A profound observation about COPD is that patients with end-stage disease, even years after they discontinue smoking, have ongoing intense airway inflammation.[51–53]

The traditional theory regarding the generation of emphysema is that there is an abnormal balance between proteases and antiproteases in the lungs of patients who develop smoking-related COPD.[54–56] That theory proposes that neutrophils accumulate in the lungs in response to inhaled particulates and become activated and produce elastase. Excess elastase overwhelms resident antiprotease reserves, causing lung destruction.[57] The neutrophil-driven, protease-antiprotease theory of the disease gains support from the occurrence of emphysema in patients with α_1-antiprotease deficiency.

Recent observations, however, have expanded our understanding of other cellular contributors to lung inflammation. "Knockout" mice deficient in macrophage elastase do not develop emphysema when exposed to cigarette smoke. In contrast, mice deficient in neutrophil protease develop emphysema when exposed to similar conditions.[58,59]

Morphologic studies further support a role for macrophages. Airway mucosal biopsies and surgical specimens from COPD patients have more macrophages and a paucity of neutrophils, even though there are more neutrophils in bronchoalveolar lavage (BAL) fluid from those patients.[48,50,60,61] Macrophage numbers are also several-fold higher in BAL fluid obtained from COPD patients.[49]

Macrophages, therefore, appear to play an important role—perhaps more important than neutrophils—in causing tissue destruction in the lungs of patients who smoke. The macrophage is replete in its capacity to produce cytotoxic tissue changes by the release of proteases and oxidants.[56,62,63] The macrophage can drive the inflammatory response by releasing chemotactic factors, such as IL-8, that recruit neutrophils to inflammatory zones.[48] Neutrophils may rapidly transit from the vascular space into the airways, which explains the paucity of those cells in tissue but their increased numbers in alveoli.[64,65]

Emphysematous lungs also contain increased numbers of lymphocytes in tissue around peripheral and central airways, and the quantity of lymphocytes correlates with the severity of emphysema.[60,61,66] The majority of lymphocytes are CD8+ T cells, as compared to CD4+ T cells, which is the inverse relationship of that in the lungs of asthmatic patients.[60,61] Cytotoxic T cells may actively damage the lung, as suggested by the occurrence of accelerated lung injury with physiologic features of COPD in individuals seropositive for human immunodeficiency virus, which

appears to be mediated by T cells.[67–69] Cytotoxic T cells have the capacity to cause apoptosis and destruction of alveolar wall epithelial cells through their release of TNF-α and perforins.[70]

Other cells with the potential to damage lung tissue include alveolar cells, epithelial cells, and eosinophils. Lung specimens taken during lung volume reduction surgery on patients with severe emphysema contain alveolar cells that express matrix metalloproteinases (MMP-1) capable of damaging lung tissue.[71] Also, epithelial cells exposed to particulates release inflammatory mediators such as IL-8 and chemokines that can recruit leukocytes.[72,73] Eosinophils play an important role in promoting tissue inflammation in asthma, but the evidence that they contribute to COPD is limited. Eosinophils are not increased in the sputum of COPD patients, but expectorated sputum contains increased concentrations of eosinophil cationic proteins and eosinophilic peroxidase.[74]

These observations indicate that we should revise our traditional theories of the primacy of the neutrophil in causing COPD and consider the importance of macrophages, lymphocytes, and possibly other cell types. The primary importance of elastase is also being reconsidered. Macrophages produce an array of proteases, including gelatinase B (MMP-9), macrophage metalloelastase (MMP-12), collagenase (MMP-1), and cathepsins S and L.[56,75,76] Each of those proteases, in addition to neutrophil elastase,[77] has the capacity to damage lung tissue. Their potential role is suggested by the increased levels of gelatinase B (MMP-9) in BAL fluid from patients with emphysema.[78] The relative importance of the various elastases may differ depending on the stage and severity of COPD.[71] Although, neutrophil elastase remains the likely mechanism in patients with α_1-antitrypsin deficiency, it may not be the most important protease in COPD that affects patients who smoke.

Evidence for a pathogenetic role of TNF-α is especially strong, because it is elevated in the airways of smokers only in the presence of COPD, which is in contrast to the elevated neutrophils and IL-8 found in airways of smokers with or without COPD.[48] Also, inhaled TNF-α promotes in humans an influx of neutrophils into airways[79] and enhances extracellular elastolytic activity by adherent neutrophils.[80]

As stated at the onset, the locus of airway obstruction in COPD is not entirely understood, with suspicion arising that inflammatory changes in small airways may play a role. A recent study that used an ultra-thin bronchofiberscope found that epithelial cells harvested from small airways had a higher expression of transforming growth factor β (TGF-β) than did controls.[45] That finding suggests that small airway epithelial cells might contribute to obstructive changes found in COPD.

Other factors potentially operative in generating inflammatory lung destruction include increased oxidant stress,[81,82] as shown by the presence of increased concentrations of hydrogen peroxide in patients' exhaled breath condensates during COPD exacerbations.[83]

Even though the development of COPD clearly depends on an inflammatory response to inhaled particulates, noninflammatory mechanisms of lung injury may also occur. Cigarette smoke produces apoptosis of microvascular cells and macrophages before the onset of tissue inflammation and damage to parenchymal extracellular matrix.[84,85] Apoptosis in epithelial and endothelial cells may occur from a loss of signaling to cell receptors, induced by inhaled cigarette smoke.[86]

The presence of damaged airways in COPD produces a bronchial milieu conducive to colonization by bacterial pathogens. It appears that airway bacteria may contribute to the progression of COPD in a "vicious circle" of inflammation.[87,88] Evidence of the role of bacterial infections in the pathogenesis of COPD derives from observations of nonsmokers with reduced FEV_1 values and who experienced frequent lower respiratory tract infections in childhood.[89–92] The mechanisms by which chronic infection might cause COPD are unclear but may relate to ongoing inflammation, with damage to the airway epithelial cells and impairment of mucociliary clearance, which promote further persistence of airway infection.[88]

Viral infections may also contribute to lung damage by priming airway inflammation. Epithelial cells that latently express adenoviral protein E1A have an enhanced capacity for the transcription of IL-8 and intercellular adhesion molecule-1 (ICAM-1), which amplify the inflammatory response to cigarette smoke, allergens, and endotoxins.[93,94] Recent attention has focused on hepatitis C virus as a viral candidate for the generation of chronic lung injury, in consideration of its putative role in the production of other chronic conditions, such as cardiomyopathy[95] and glomerular disease.[96] BAL fluid from patients with chronic hepatitis C virus infection shows increased numbers of lymphocytes, suggesting that hepatitis C virus infection promotes a lymphocytic alveolitis, which might progress to chronic lung disease.[97]

Natural History

COPD patients experience a long preclinical course, followed by a slow progression of measurable deterioration in air flow, punctuated by acute exacerbations of airway disease (Fig. 5). Air flow obstruction progresses uniformly in most patients, although some experience relatively steep declines, with loss of lung function occurring in discrete stages (see Fig. 5). As shown by the National Heart, Lung, and Blood Institute Lung Health Study, smoking cessation is the only intervention that can slow

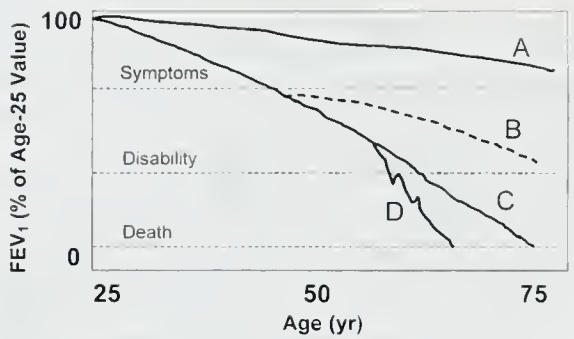

Fig. 5. Natural history of chronic obstructive pulmonary disease (COPD) as measured by forced expiratory volume in the first second (FEV_1) as a percentage of the baseline value at age 25. Normal nonsmoking individuals (A) have a progressive loss of FEV_1 but never become symptomatic with airway obstruction. Patients with COPD who quit smoking (B) experience an FEV_1 decline that parallels a nonsmoking, age-matched person. Patients with progressive COPD (C) can develop a loss of FEV_1 that may eventually produce symptoms, disability, and death. Some COPD patients (D) have steep declines in lung function during discrete clinical episodes.

the progression of airway obstruction experienced with COPD.[98]

Predicting the decline of lung function in individual patients is difficult because of the variable course of the disease and the lack of studies that examine individual predictors. Population studies indicate that heavy current and lifetime smokers experience a more rapid decline than average smokers.[98] Recent studies identify increased airway responsiveness to inhaled methacholine as a predictor of accelerated decline in lung function.[99] Chronic mucus hypersecretion can affect the natural history of COPD, being associated with a more rapid decline in FEV_1 and higher risk of hospitalization.[100,101] Other predictors of a poor clinical course include low functional status,[102] polymorphism in the TNF-α gene promoter region,[103] and elevated levels of fibrinogen.[104]

Otherwise, little is known regarding predictors of lung function decline in individual patients. Moreover, little is known regarding other aspects of the natural history of COPD in general populations of patients since the modern era of treatment. Previous studies followed patients with severe COPD and limited the observations to measures of lung function.[36,105,106] Studies have not comprehensively examined the impact of COPD on health-related quality of life, comorbid conditions, or predictors of mortality. We are learning, however, that low body mass index is a marker of functional capacity, health status, and premature death, independent of the severity of air flow limitation.[107,108] Weight loss reflects loss of skeletal muscle[109] and develops as a consequence of various factors, such as increased energy requirements in excess of dietary intake, disuse atrophy, malnutrition, low levels of anabolic steroids, and myopathy from corticosteroid use.[110] A combination of oral nutritional supplements and exercise in addition to anabolic stimulus are under investigation to determine their role in reversing weight and strength loss.[110]

Acute exacerbations play a major role in the morbidity and mortality of patients with moderate to severe COPD.[111] Patients who present with advanced disease requiring hospitalization for acute exacerbations have a limited long-term prognosis, with more than 30% dying within 1 year of hospital discharge.[27] Patients older than 65 years admitted to an intensive care unit (ICU) have a 1-year mortality of 60%, regardless of the need for invasive or noninvasive mechanical ventilation.[112] Lower respiratory tract infections are the most common cause of death.[111]

The presence of COPD also increases the risk of lung cancer over and above the risk related to smoking.[98]

Etiologic Factors

Cigarette smoking remains the most important determinant of the development and accelerated progression of COPD, being found in 85–90% of COPD patients.[113] Greater evidence now exists, however, that COPD can occur from certain occupational inhalational exposures and from particulate air pollution.[114]

Recent studies have identified other contributory factors. Inadequate treatment of chronic asthma can lead to irreversible air flow obstruction that is a part of the spectrum of COPD.[19,115] The Copenhagen City Heart Study[116] followed men and women older than 20 years and found that socioeconomic status, based on income and education, was associated with decreased lung function, independent of smoking history.[116] The association of socioeconomic index with lung function is unclear but may relate to childhood infections, inhalation exposures in the environment, occupational exposures, or poor housing.

Other factors include differences in genetic susceptibility to the cytotoxic effects of inhaled particulates.[117,118] Familial aggregations of severe early-onset COPD[119] and differing prevalence of COPD among ethnic groups[120] support a role for genetic susceptibility.

Diagnosis

COPD develops in an insidious manner, with most patients experiencing, at the onset of clinical disease, mild or nonspecific symptoms to which they attach little importance.[98,121,122] Most patients are diagnosed when they present with acute exacerbations of airway disease.

Even when early symptoms are recognized, their nonspecific nature often fails to suggest to physicians a diagnosis of COPD.[123,124] Although most COPD patients have increased cough and sputum production, the converse is not true. The presence of respiratory symptoms requires

confirmation of air flow limitation to establish the presence of COPD. Because COPD is defined in physiologic terms, pulmonary function testing is required to diagnose the disease.

Unfortunately, pulmonary function testing is under-utilized in evaluating patients with nonspecific respiratory conditions. A study of patients in a university ambulatory health care system and a health maintenance organization found under-utilization of spirometry for patients with respiratory symptoms compatible with COPD.[125] Only 42% of the university patients and 33% of the HMO patients underwent spirometry, as compared with 64% and 50% who underwent electrocardiography. In the NHANES III study over 80% of subjects with respiratory symptoms had visited a physician during the previous 12 months but did not undergo spirometry.[32] The under-diagnosis of COPD appears to affect women more than men.[126]

Because of the importance of detecting early disease, incorporation of spirometry into routine health care screening, regardless of smoking history, has been suggested. In Holland, the recently completed Detection, Interaction, and Monitoring of COPD and Asthma Project screened a randomly sampled population of people 25–70 years of age for air flow limitation defined by FEV_1 criteria, symptoms, or bronchodilator reversibility.[127] The investigators determined that 52% of screened persons had possible airway disease, which included patients with possible asthma in addition to COPD. The investigators followed their sample to detect individuals who suffered rapid decline in lung function. The cost of detecting such patients was $500–$1000 per person, which compares favorably with other health care screening interventions.

The GOLD consensus experts doubted that spirometric screening is sufficiently sensitive and cost-effective to recommend for general health care screening, and they proposed spirometry for patients with the onset of respiratory symptoms compatible with COPD.[16] Limiting screening to current smokers with respiratory symptoms, however, will overlook a majority of the population with air flow obstruction and miss opportunities to lessen morbidity and mortality.[33] The National Lung Health Education Program is recruiting primary care physicians in the diagnosis of COPD by promoting the widespread use of simple spirometry to detect air flow limitation.[128,129] They recommend spirometry for patients older than 45 years and with a smoking history and for any person with respiratory symptoms.[130] The program's public message is "Test Your Lungs, Know Your Numbers."[128]

Pulmonary function testing also serves to stage the severity of COPD and characterize airway reactivity.[18] The ATS and ERS staging systems both use FEV_1. The British Thoracic Society system also uses clinical features of cough, sputum, dyspnea, and lung sounds. Spirometry assists in educating patients about their disease. Although many practitioners remain reluctant to use spirometry, it has the added benefit of identifying patients at highest risk of overall mortality and death from ischemic heart disease and stroke.[131] The higher risk of cardiac disease associated with COPD may be related to the increased serum fibrinogen levels of COPD,[104,132] which are predictors of increased cardiovascular risk.

Patient Monitoring

Despite the importance of spirometry for establishing the presence of disease, spirometry findings do not correlate well with the severity of respiratory symptoms or patients' overall health status.

Other measures are increasingly being used to monitor the clinical course of COPD patients. Because spirometric response to bronchodilators may underestimate bronchodilator benefit, measures of health-related quality of life and functional capacity are increasingly being used to determine bronchodilator response, in lieu of physiologic measures.[133,134]

Health-related quality of life refers to the physical, psychological, income, freedom, and social domains of health that are unique to each person.[135,136] Patients find disease-specific health-related quality of life measurements more relevant to their medical problems than physiologic measurements. Health-related quality of life measurements are also more responsive and have a greater potential to detect important and meaningful change from treatment interventions.[137] Studies of medical therapies for COPD are increasingly including health-related quality of life measures to determine outcomes.[138]

Additional measurements used to monitor the course of COPD patients include the number of airway exacerbations and hospitalizations, relief of symptoms, inhibition of long-term lung function decline, increase in performance status, and increase in life expectancy.

Pharmacologic Therapy of Stable Disease

The treatment of COPD is symptomatic, because no intervention has been found to alter the progression of air flow limitation. Some interventions, such as vaccination for influenza and pneumococcal pneumonia, and continuous oxygen therapy for hypoxic patients, have been shown to improve life expectancy.

Bronchodilator drugs represent the cornerstone of COPD therapy. Although one third of patients have no measurable spirometric response to the inhalation of bronchodilators, bronchodilator therapy still provides many patients with symptomatic relief from fixed airway obstruction and improved functional capacity.[18,139,140]

Short-acting β-agonist bronchodilators have served as traditional therapy because they decrease severity of cough-

ing and reduce breathlessness by improving lung emptying through bronchodilation. Short-acting drugs also decrease histamine release, decrease airway hyperresponsiveness, improve diaphragmatic contraction, inhibit inflammatory response, and enhance ciliary function.[141,142]

The advent of long-acting β agonists and anticholinergic agents, however, has relegated short-acting β agonists to rescue therapy. Recent research indicates that regular use of short-acting agents results in twice as much drug use, without any physiologic or clinical benefit, as on an as-needed basis.[143]

Long-acting β agonists such as salmeterol have supplanted short-acting drugs for long-term maintenance therapy. Salmeterol not only improves lung emptying and severity of coughing but also improves health status and prolongs intervals between exacerbations.[133,144] Salmeterol can produce bronchodilation even for patients who are unresponsive to albuterol. Salmeterol also has anti-inflammatory properties in decreasing adhesion of bacteria to airway epithelial cells.[145]

Salmeterol owes its long duration of action to an interesting property of binding to the active site and producing repeated receptor stimulation. Because of its lipophilic properties, it diffuses to reach the β_2 adrenoreceptor through the cell membrane.[146] The drug's side chain binds to a discrete hydrophobic domain within the exosite of the receptor, which prevents dissociation of the drug. The saligenin head of the molecule is free to engage and disengage with the active receptor site in a hinge-like manner.[147]

Formoterol is another long-acting β agonist. It is delivered twice a day and has a faster onset of action than salmeterol,[148,149] but the mechanisms of its prolonged action are unknown.[150] Although it appears to be as effective and safe as salmeterol when prescribed long-term for asthma patients,[151] no long-term safety or efficacy data are available for formoterol in COPD. When administered in its inhaled, dry powder form it has been found to have a greater effect than ipratropium in improving symptoms and quality of life during 3 months of therapy.[152] Salmeterol and formoterol have similar bronchodilator effects for patients with a range of COPD severity,[149] although 1 study found greater efficacy with salmeterol for patients with severe disease airway obstruction.[153] Another recent study found that combination therapy with formoterol and ipratropium is more effective than a combination of salbutamol and ipratropium, with regard to functional and spirometric measurements.[154]

The bronchodilator effects of both salmeterol and formoterol remain stable with regular use, in contrast to the diminished efficacy observed with long-term use of short-acting β agonists.[155]

Ipratropium bromide has been considered first-line therapy for COPD patients and provides a range of benefits.[17,18,25,156] During acute episodes of breathlessness it has bronchodilation properties equal to β agonists.[157] Ipratropium also reduces mucus hypersecretion, hyperinflation, dyspnea, and episodes of nocturnal oxygen desaturation; it also improves sleep quality and exercise tolerance.[144,158] Ipratropium has been shown in some[144,159] but not all[152] studies to improve quality of life and decrease the frequency of acute exacerbations of COPD. In contrast to short-acting β agonists, ipratropium produces sustained effects without tachyphylaxis.[139] Ipratropium provides additive effects in improving FEV_1 when combined with salmeterol or formoterol for COPD patients.[148,160,161] Ipratropium has been shown to be safe in elderly COPD patients.[162] Recent studies showing greater efficacy with long-acting β agonists[144,152,163] challenge the first-line position of ipratropium in treating COPD.

Although not approved for use in the United States, tiotropium is a long-acting, once-daily, inhaled anticholinergic drug. The onset of action occurs within 30 min and effects persist over 24 hours.[164,165] Existing studies indicate that tiotropium improves lung function to a greater degree than ipratropium, with a similar safety profile.[164–166]

Although published guidelines state that theophylline benefits some COPD patients,[18] its narrow therapeutic index and weak bronchodilator effects limit its use. Added potential benefits include enhanced right ventricular function, dyspnea relief, improved mucociliary clearance, increased diaphragmatic function, and increased exercise tolerance.[167,168] The combination of theophylline and salmeterol has greater effects than either drug alone.[169] We have learned, however, to reserve aminophylline for persistently symptomatic patients already treated with β agonists and who benefit from the addition of aminophylline.

Although long-term systemic corticosteroid drugs have been used for COPD patients, recent data do not support their role. Most studies have not shown reduction of inflammatory mediators, neutrophil-mediated inflammation, or protease activity in patients treated with steroids.[170,171] although a recent study indicates that neutrophilic inflammation in sputum from patients with stable COPD may be decreased with inhaled corticosteroids.[172] In vitro, corticosteroids have been shown to prolong rather then diminish neutrophil survival.[173]

Long-term use of systemic corticosteroids by patients with stable COPD causes myopathy, which worsens ventilatory and functional capacity and decreases survival.[174] At present, long-term treatment with corticosteroids is not recommended for COPD patients.[175–177] Although a short course of systemic corticosteroids may improve spirometric measures of air flow limitation, this improvement has no consistent correlation with enhanced functional capacity, quality of life, exercise tolerance, or risk of exacerbation. Also there is no evidence to support the role of a short course of oral corticosteroid to predict responsiveness to inhaled corticosteroids.[178,179]

As systemic corticosteroids wane in their role for managing COPD, intense interest has focused on inhaled corticosteroids. The inflammatory nature of COPD is the rationale for their use. An anti-inflammatory effect of inhaled corticosteroids is supported by observations that exhaled nitric oxide levels are decreased in patients treated with inhaled beclomethasone.[180] Also, observational, population-based studies indicate that elderly COPD patients who receive inhaled corticosteroids after a hospitalization have 24% fewer repeat hospitalizations for COPD and lower all-cause mortality.[181]

Since 1995, 5 prospective, randomized studies have examined the utility of inhaled corticosteroids in patients with various severities of stable COPD.[121,178,182–184] In aggregate, those studies show that inhaled corticosteroids do not alter the natural history of COPD by slowing FEV_1 decline. However, the ISOLDE (Inhaled Steroids in Obstructive Lung Disease) study found that inhaled fluticasone slows the rate of decline in disease-specific measures of health status and reduces the risk of clinical exacerbations in subsets of patients.[178,185] The Lung Health Study found that inhaled triamcinolone may attenuate airway hyperresponsiveness and reduce clinical symptoms of COPD, including dyspnea and cough, and decrease unscheduled physician visits and hospitalizations.[182]

Those studies show modest benefit from inhaled corticosteroids for COPD patients. That benefit is further supported by a recent study that found that elderly patients with severe COPD experienced deterioration in ventilatory function and increased exercise-induced dyspnea after withdrawal of inhaled corticosteroid therapy.[186] Consequently, the GOLD guideline recommends inhaled corticosteroids for symptomatic COPD patients with $FEV_1 < 50\%$ of predicted and repeated exacerbations that require treatment with antibiotics or systemic corticosteroids.[16]

Unfortunately, the dose-response relationship and long-term safety of inhaled corticosteroids are not known. Inhaled triamcinolone ($\geq$ 400 μg/d) for premenopausal women causes a dose-related decline in bone density, both at the total hip and trochanter, and increased bruising.[187]

Advances in Surgical Therapy

During the last 20 years, lung transplantation has moved from being an experimental procedure to being a standard therapy. It offers relief of disabling symptoms for patients with end-stage COPD, although it does not increase life expectancy.[188] From 1985 to 1999, nearly 10,000 single or double lung transplantations were performed, in 153 lung transplantation programs.[189] Criteria for transplantation are based on an $FEV_1 < 25\%$ of predicted (without reversibility). $P_{aCO_2} > 50$ mm Hg, and the presence of secondary pulmonary hypertension.[190] Lifetime expenditures for lung transplantation range from $110,000 to well over $200,000.[191,192] The major limitation of the procedure is the lack of suitable organ donors. Ten to 15% of patients die while on the waiting list, which is typically 2 years long until a transplantation is received.[193]

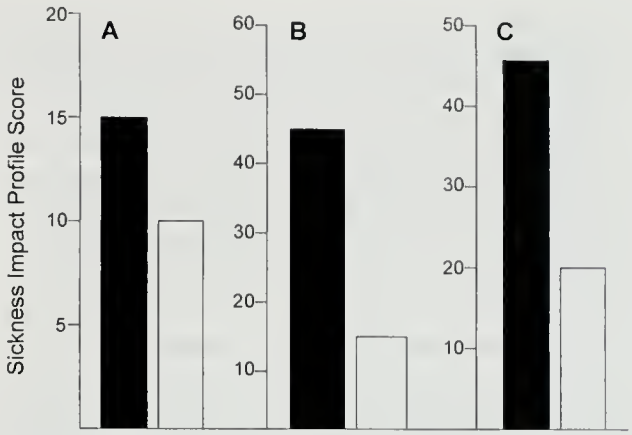

Fig. 6. Comparison of total Sickness Impact Profile score (A) and components of physical (B) and psychological (C) scores before (black bars) and 3 months after (clear bars) lung volume reduction surgery (n = 42). (Adapted from Reference 210.)

Lung volume reduction surgery (LVRS) provides palliative relief for selected COPD patients, although, like lung transplantation, a survival benefit has not been established. Several ongoing trials are examining the benefits of LVRS and identifying selection criteria.[194] Patients with severe COPD ($FEV_1 < 20\%$ of predicted), homogeneous emphysema, or a very low diffusing capacity ($< 20\%$ of predicted) are at high risk of death from the procedure and appear unlikely to benefit.[195]

Results to date indicate that LVRS improves pulmonary mechanics, functional capacity, weight gain, and quality of life.[196–210] Quality of life improvements appear related to the degree that surgery reduces hyperinflation rather than the degree of improvement in FEV_1 (Fig. 6).[210] LVRS improves lung function by increasing lung elastic recoil and enhancing respiratory muscle function with improved diaphragmatic mechanics and improved inspiratory respiratory muscle and increased neuromechanical coupling, irrespective of changes in FEV_1.[204–209] FEV_1 is not a good marker of outcome because it correlates poorly with dyspnea.[137] Patients with the most severe baseline emphysema experience the greatest improvement in exercise capacity after LVRS.[211] For patient selection, computed tomography morphometry can separate lungs into compartments of severe, mild-to-moderate, and normal, thereby predicting the response to LVRS.[211] Other predictors of benefit from LVRS include more severe emphysema, upper lobe predominance, greater heterogeneity, and a volume of normal attenuation lung (measured by computed tomography) of > 1 L.[212]

The benefits of LVRS deteriorate over time, but some patients can retain benefit for 2–5 years.[196–201] The total hospital cost of LVRS ranges from $11,712 to $121,829, with a mean cost of $30,976 (median $19,771).[213]

Most patients with emphysema are poor candidates for either lung transplantation or LVRS. Ideal candidates for LVRS are patients with hyperinflation, heterogeneous distribution of disease, FEV_1 of > 20% of predicted, and normal P_{CO_2}.[214]

Nocturnal Noninvasive Ventilation

Patients with severe COPD have mechanically disadvantaged and chronically fatigued respiratory muscles. Consequently it would appear likely that nocturnal noninvasive positive pressure ventilation (NPPV) would benefit those patients by unloading respiratory muscles. Unfortunately, recent studies do not support a role for nocturnal NPPV in stable COPD patients. Although one study found improved quality of life with nocturnal ventilation,[215] end points were measured at only 3 months. In studies with 1-year follow-up, nocturnal NPPV did not alter the course of the disease and provided only marginal benefit.[216] The aggregated studies do not support a role for nocturnal NPPV for stable patients with severe COPD.[217,218]

Rehabilitation

Pulmonary rehabilitation represents one of the earliest examples of a disease management program that combines inpatient-to-outpatient transitional care and ambulatory care directed to enhance adaptation to disease and quality of life. Unfortunately, pulmonary rehabilitation has been under assault from payer organizations—most notably Medicare—despite recent confirmation of program effectiveness.

The primary purpose of pulmonary rehabilitation is to reduce respiratory-related symptoms, improve quality of life, and promote physical and emotional participation in activities of daily living.[219,220] Patient and family education combined with breathing and exercise training are keystones of rehabilitation programs. Recent studies show that patients respond to enrollment in rehabilitation with increased exercise tolerance and decreased symptoms of fatigue and dyspnea,[221] with sustained benefits after program completion.[222,223] Quality of life and cognitive function are also improved for patients with moderate-to-severe COPD, for as long as 2 years after completion of ambulatory rehabilitation.[202,224–226] Rehabilitation also decreases the amount of health care needed, with decreased unscheduled physician visits, decreased use of medications, fewer exacerbations, and shorter hospital stays when hospitalized.[202,224] Repeat consolidation enrollments at yearly intervals provide similar short-term gains but do not provide additional physiologic or functional benefit.[224]

Pulmonary rehabilitation can also provide end-of-life education to COPD patients who have progressive disease that can result in respiratory failure and an unexpected need for life-supportive interventions.[227–229] This education can promote advance care planning, patient-physician discussions about end-of-life care, and patient confidence that the physician understands his or her end-of-life wishes.[230] Such education is well-accepted by patients and does not provoke troubling anxieties.[231] Unfortunately, most pulmonary rehabilitation programs do not present advance care planning to their enrolled patients.[232]

Acute Exacerbations of Chronic Obstructive Pulmonary Disease

Acute exacerbations have a profound effect on patients' quality of life.[233] Most COPD patients have 2 or 3 exacerbations per year, with each exacerbation lasting 12 days.[234] Although there is no generally accepted definition of an acute exacerbation, most definitions contain elements of worsening dyspnea, increased sputum purulence, and increased sputum volume. Definitions differ, however, between the COPD consensus statements of professional societies.[17,18] The recent evidence-based report from the American College of Chest Physicians (ACCP) and American College of Physicians/American Society of Internal Medicine (ACP-ASIM) defines an acute exacerbation as the occurrence of "some combination of 3 clinical findings: worsening of dyspnea, increase in sputum purulence, and increase in sputum volume."[235]

The severity of exacerbations is staged by the "Winnipeg criteria."[236] Type 1 (severe) exacerbations include all 3 of the following: increased dyspnea, sputum volume, and sputum purulence. Type 2 exacerbations include any 2 of those 3 conditions. Type 3 (mild) exacerbations include any 1 of those 3 conditions, with the addition of an upper respiratory infection within the past 5 days, fever with no other cause, increased wheezing or cough, or a respiratory rate or heart rate 20% above baseline.[236]

Acute exacerbations are accompanied by signs of increased airway inflammation. Expectorated sputum contains higher concentrations of neutrophils, interleukin (IL)-6, and IL-8 than stable patients.[237] During periods of stability, patients who experience frequent exacerbations have higher levels of interleukin 6 in sputum than do stable patients with few exacerbations.[237] It is becoming increasingly apparent that COPD patients assume an allergic profile of airway inflammation during acute exacerbations. Bronchial biopsies from patients with mild COPD show increased infiltration of eosinophils during exacerbations.[47,64,238,239] This influx of eosinophils may result from the upregulation of RANTES (regulated upon activation,

normal T cell expressed and secreted) in the epithelium and subepithelium that has been observed during acute exacerbations.[240]

Common etiologies of acute exacerbation include airway infection,[241,242] particulate air pollution,[243] and environmental temperature changes.[244] The etiology is not identified in one third of acute exacerbations.[245] The etiologic role of those factors has only recently been confirmed, and the pathogenesis of acute exacerbations remains incompletely defined. Considerable progress has been made, however, in the last 5 years in understanding both etiology and pathogenesis.

Fifty years ago an association between respiratory illnesses and pollutants was suspected when more respiratory deaths were noted during episodes of "London fog," which was composed of acid aerosols, sulfur dioxide, and black smoke pollutants.[246] More recently, epidemiologic studies established a clear link between particulate air pollution and higher respiratory morbidity.[247–250]

Persons with COPD appear to be "primed" to develop airway inflammation when exposed to air pollutants, because of abnormalities in mucociliary clearance, more macrophages in alveolar spaces, and greater deposition of particulates into small airways than individuals with healthy lungs.[251] Air pollution increases airway mucus, which cannot be readily expectorated, because of impaired mucociliary function.[252] Retained mucus may increase airway resistance and plug small airways, which is a common finding at autopsy of COPD patients who die from acute respiratory failure. Epithelial cells stimulated by inhaled particulates release proinflammatory mediators, such as IL-8 and the chemokine RANTES, which promote an influx of inflammatory leukocytes.[72,73] Airway macrophages release IL-8 and TNF-α in response to their phagocytosis of inhaled particulates. Ultrafine particles ($<$ 14 nm in cross-sectional diameter) have direct cytotoxic effects on the lung and stimulate an inflammatory response with an influx of leukocytes.[253] Particulates that contain transition metals produce oxidative stress and promote the movement of transcriptional activator nuclear factor-kB to the nucleus to promote the expression of proinflammatory genes.[254]

Bronchial infections are a major cause of acute exacerbations of COPD. The airway epithelium serves as a protective barrier against external irritants that would invade lung tissue, and epithelial membranes promote clearance of inhaled particulates through mucociliary mechanisms. Bronchial infections disrupt normal epithelial functioning[145,255] and induce epithelial cells to release proinflammatory mediators. Bacterial, atypical bacterial, and viral infections are the 3 categories of bronchial infection associated with acute exacerbations.

Bacteria (*Haemophilus influenzae*, *Moraxella catarrhalis*, and *Streptococcus pneumoniae*) can be isolated from the sputum of 40–60% of COPD patients during acute exacerbations.[256] Some suggested that these bacteria are "innocent bystanders" or simple colonizers,[257] because a causal link between airway bacteria and acute exacerbations had not been established. Recently, however, studies using bronchoscopic sampling of the lower respiratory tract showed that 50% of patients with acute exacerbations have bacterial infections in distal airways.[258–261] The concentration of bacteria in small airways is higher in patients suffering acute exacerbations than in patients with stable COPD.[259] Serologic studies to establish a causal link by identifying an immune response to bacteria were limited by the previously available techniques that determined antibody responses to whole bacteria.[262] However, a recent longitudinal study by Murphy and Sethi examined COPD patients and used antibodies specific to surface-exposed epitopes.[263] They observed a specific immune response for the infecting strain of *H influenzae*, which established a causal role for bacterial infection in acute exacerbations. Measures of airway inflammation also correlate with evidence of airway infections during exacerbations.[256]

Uncertainty exists as to why bacterial pathogens chronically harbored in the airways of COPD patients only occasionally cause bronchial infections. Molecular epidemiology studies recently showed that there is a dynamic turnover of different bacterial strains in the lower airways and that the introduction of a new strain corresponds with an acute exacerbation.[87,256] That observation suggests that patients suppress bacterial growth in their lower airways with specific immune response. The introduction, however, of a more virulent strain or a new strain for which a specific immune response does not exist causes infection and acute exacerbation. Consequently, measuring the bacterial load in the airways of a COPD patient does not assess the risk for an acute exacerbation.[87,256]

Atypical pathogens also contribute to acute exacerbations, but less frequently than the major pathogens listed above. *Mycoplasma pneumoniae* is a rare cause of acute exacerbation.[258] *Chlamydia pneumoniae* is associated with 5–10% of acute exacerbations.[264] In patients admitted to the ICU with severe COPD, however, *C pneumoniae* may account for 18% of admissions, but usually in combination with other bacteria.[258]

Viruses have long been known to precipitate acute exacerbations of COPD and account for up to 30% of episodes.[265] Viral bronchitis is present in 16% of COPD patients admitted to the ICU for mechanical ventilation.[258] Many patients with viral infections, however, have concomitant bacterial infections. As previously mentioned, viruses have a curious capacity to produce latent infections by intercalating a portion of their genome into lung tissue, which can augment the inflammatory response to subsequent injury.[266]

The prognosis of patients who require hospitalization for acute exacerbations is guarded. More than 50% of patients hospitalized for acute exacerbations of COPD require readmission within the next 6 months,[27,267] and 3–4% of hospitalized patients die.[268] Mortality is 11–24% for those patients admitted to the ICU.[27,112,269] The 1-year mortality of patients who survive an ICU stay is 43–46%.[27,112,269]

Because of this guarded prognosis, investigations have focused on identifying high-risk patients. Predictors of need for hospitalization of patients with acute exacerbations include previous admissions, lower FEV_1, and under-prescription of long-term oxygen therapy.[270] Once hospitalized, FEV_1 at admission and over the first several days of hospitalization is highly predictive of clinical outcome.[271] For intubated patients the presence of comorbidities and measures of acute illness severity are predictors of survival.[272] Extubation failure or the need for mechanical ventilation beyond 72 hours also portends a worse prognosis for survival.[272] Unfortunately, most models are not validated, and mortality models do not successfully identify patients with > 90% likelihood of dying.[273] Models predicting relapse have only a 57% sensitivity and a 72% specificity.[274]

Recommendations for managing acute exacerbations are limited by the paucity of prospective randomized studies. The recently published conjoint ACCP/ACP-ASIM guideline for management of acute exacerbations[235,273,275] based its care recommendations on a comprehensive evidence-based report sponsored by the Agency for Healthcare Research and Quality. Many of the therapeutic recommendations in that guideline, however, depend on consensus derived from incomplete investigative data.

The ACCP/ACP-ASIM guideline reports that 14 randomized trials support the conclusion that short-acting β agonists and anticholinergic-type inhaled bronchodilators have similar efficacy and that they both have a greater effect than parenteral bronchodilators on spirometry values.[235,273] Concerns about toxicity relegate aminophylline to being a second-line bronchodilator. No differences exist in the delivery of inhaled bronchodilators via metered-dose inhaler or nebulizer.[235,273]

Six randomized, placebo-controlled studies now support the utility for hospitalized patients of a short course of corticosteroids, which improves spirometry values and decreases relapse rates.[235,273] Improvements in FEV_1, however, are transient and subside by day 4 of hospitalization.[276] Unfortunately, trials to date have not established the ideal dose or duration of glucocorticoid therapy for hospitalized patients. It has become clear, however, that 2 weeks of therapy is as good as 8 weeks (Fig. 7).[277] The Systemic Corticosteroids in COPD Exacerbation (SCCOPE) study used 125 mg intravenous solumedrol 4 times a day for 72 hours, followed by a tapering dose of prednisone over 2 weeks.[277] The GOLD guideline recommends 30–40 mg prednisone a day for 10–14 days.[16] There are few data regarding the use of corticosteroids in the outpatient setting for acute exacerbations.

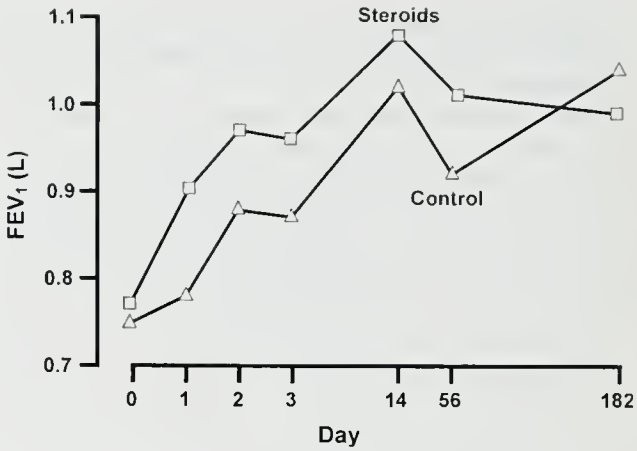

Fig. 7. Forced expiratory volume in the first second (FEV_1) in patients hospitalized for acute exacerbation of chronic obstructive pulmonary disease (COPD). Patients treated with steroids had a more rapid improvement in FEV_1 than did patients treated with placebo, but the differences were no longer statistically significant after day 4. (Adapted from Reference 277.)

Antibiotics most likely benefit acute exacerbations and the ACCP/ACP-ASIM guideline recommends their use.[273] Most studies that examined the utility of antibiotics were completed before our era of resistant pathogens. Antibiotic selection and duration of therapy remain unclear. Patients with Type 1 exacerbations appear most likely to benefit from antibiotic therapy.

Other treatment modalities, such as mucus clearance strategies with expectorants, mucolytic agents, and mucokinetics, have no evidence of benefit for shortening disease course but may provide some benefit in improving symptoms.[273] Physical therapy during an acute exacerbation is not beneficial and may present risks of functional deterioration.[273]

Noninvasive Positive-Pressure Ventilation for Acute Exacerbations

Five randomized controlled trials have shown that NPPV benefits subgroups of hospitalized patients with acute exacerbations, by decreasing the need for intubation, shortening hospital stay, and improving survival.[278–282] Up to 20% of hospitalized COPD patients may be candidates for NPPV.[283] A more recent study indicates efficacy for COPD patients (hospitalized on wards) who have mild acidosis.[284] The benefits include a lower risk of pneumonia than with invasive ventilatory support.[285] NPPV also serves as a support for patients who fail extubation.[286,287]

As reviewed in a recent ATS consensus conference, NPPV benefits patients by unloading respiratory muscles and augmenting alveolar ventilation until the patient's underlying airway problems can reverse.[288] Unfortunately, NPPV is not widely available for managing acute exacerbations. In Great Britain only 48% of hospitals have the capacity for NPPV and 68% do not treat more than 20 patients per year.[289]

Future Directions

Although the above discussion illustrates our progress in understanding COPD, I omitted multiple topics that establish new frontiers in caring for patients. We have gained in our understanding of the physiologic effects of nicotine that underlie its addicting potential,[290] the pathophysiology of nicotine withdrawal,[291] and the genetic basis of nicotine addiction.[292–294] We have gained in knowledge of interventions for smoking cessation, with the discovery that the combination of bupropion and the nicotine patch results in 36% remaining cigarette-free at 1 year.[295] We have learned extensively about the interactions of COPD and sleep-related respiratory disturbances.[296,297] And most exciting are discoveries in the field of lung repair. Recent reports indicate that treatment with retinoic acid reverses emphysema in rat models.[298,299]

Recent studies have established COPD as a highly prevalent condition worldwide, with profound impact on world health. Unfortunately, we have also learned that COPD is an under-diagnosed condition, long neglected by public health leaders and clinical investigators. COPD receives disproportionately less money from the National Institutes of Health than other illnesses, when indexed for mortality, disability, and years of life lost.[300] Fewer than 1,100 patients have been enrolled in randomized, placebo-controlled trials of antibiotics, fewer than 650 patients have been enrolled in studies comparing corticosteroids with placebo, and no controlled trials have enrolled patients with milder outpatient exacerbations.[273] These observations, however, have ushered in an era—for the first time—of national public health campaigns for educating the public and physicians about COPD.[128,300]

We can be encouraged by our new knowledge that COPD is an inflammatory disorder. Our expanded understanding of the role of cytokines and other inflammatory mediators offers opportunities to identify molecular targets for therapy and prevention of the disease.

As stated at the beginning of this review, the growth of our knowledge of COPD has been exponential, which is not only a statement of the past, but also a projection of the future. I anticipate an even greater rate of rise in our understanding of this profoundly important disorder, an understanding that will span the psychological and pharmacologic addictive properties of smoking and the nature of inflammation within the lung.

REFERENCES

1. National Center for Health Statistics. Vital Health Stat Series 1998; No. 10:193.
2. Hurd S. The impact of COPD on lung health worldwide: epidemiology and incidence. Chest 2000;117(2 Suppl):1S–4S.
3. NHLBI morbidity and mortality charthook, 2000: Available at http://www.nhlbi.nih.gov/resources/docs/cht-book.htm (accessed Feb 28, 2002)
4. Mortality patterns: preliminary data, United States, 1996. MMWR Morb Mortal Wkly Rep 1997;46(40):941–944.
5. Sullivan SD, Ramsey SD, Lee TA. The economic burden of COPD. Chest 2000;117(2 Suppl):5S–9S.
6. Hilleman DE, Dewan N, Malesker M, Friedman M. Pharmacoeconomic evaluation of COPD. Chest 2000;118(5):1278–1285.
7. Grasso ME, Weller WE, Shaffer TJ, Diette GB, Anderson GF. Capitation, managed care, and chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;158(1):133–138.
8. Wilson L, Devine EB, So K. Direct medical costs of chronic obstructive pulmonary disease: chronic bronchitis and emphysema. Respir Med 2000;94(3):204–213.
9. Murray CJL, Lopes AD. The global burden of disease. Cambridge, MA: Harvard University Press; 1996.
10. Murray CJ, Lopez AD. Evidence-based health policy: lessons from the Global Burden of Disease Study. Science 1996;274(5288):740–743.
11. Feenstra TL, van Genugten ML, Hoogenveen RT, Wouters EF, Rutten-van Mölken MP. The impact of aging and smoking on the future burden of chronic obstructive pulmonary disease: a model analysis in the Netherlands. Am J Respir Crit Care Med 2001; 164(4):590–596.
12. Silverman EK, Weiss ST, Drazen JM, Chapman HA, Carey V, Campbell EJ, et al. Gender-related differences in severe, early-onset chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2000;162(6):2152–2158.
13. Soriano JB, Maier WC, Egger P, Visick G, Thakrar B, Sykes J, Pride NB. Recent trends in physician diagnosed COPD in women and men in the UK. Thorax 2000;55(9):789–794.
14. Lacasse Y, Brooks D, Goldstein RS. Trends in the epidemiology of COPD in Canada, 1980 to 1995. COPD and Rehabilitation Committee of the Canadian Thoracic Society. Chest 1999;116(2):306–313.
15. Pauwels RA. National and international guidelines for COPD: the need for evidence. Chest 2000;117(2 Suppl):20S–22S.
16. Pauwels RA, Buist AS, Calverley PM, Jenkins CR, Hurd SS. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop summary. Am J Respir Crit Care Med 2001;163(5):1256–1276. Respir Care 2001;46(8):798–825.
17. Siafakas NM, Vermeire P, Pride NB, Paoletti P, Gibson J, Howard P, et al. Optimal assessment and management of chronic obstructive pulmonary disease (COPD). The European Respiratory Society Task Force. Eur Respir J 1995;8(8):1398–1420.
18. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. American Thoracic Society. Am J Respir Crit Care Med 1987;136(1):225–244.
19. Dow L. Asthma versus chronic obstructive pulmonary disease: exploring why 'reversibility versus irreversibility' is no longer an appropriate approach. Clin Exper Allergy 1999;29(6):739–743.

20. Gibson PG, Hargreave FE, Girgis-Gabardo A, Morris M, Denburg JA, Dolovich J. Chronic cough with eosinophilic bronchitis: examination for variable airflow obstruction and response to corticosteroid. Clin Exp Allergy 1995;25(2):127–132.
21. Gibson PG, Zlatic K, Scott J, Sewell W, Woolley K, Saltos N. Chronic cough resembles asthma with IL-5 and granulocyte-macrophage colony-stimulating factor gene expression in bronchoalveolar cells. J Allergy Clin Immunol 1998;101(3):320–326.
22. American Thoracic Society. Evaluation of impairment/disability secondary to respiratory disorders. Am Rev Respir Dis 1986;133(6): 1205–1209.
23. Canadian Thoracic Society Workshop Group. Guidelines for the assessment and management of chronic obstructive pulmonary disease. CMAJ 1992;147(4):420–428.
24. Thoracic Society of Australia and New Zealand. Guidelines for the management of chronic obstructive pulmonary disease. Mod Med Aust 1995;38:132–46.
25. BTS guidelines for the management of chronic obstructive pulmonary disease: the COPD Guidelines Group of the Standards of Care Committee of the BTS. Thorax 1997;52 Suppl 5:S1–S28.
26. Viegi G, Pedreschi M, Pistelli F, Di Pede F, Baldacci S, Carrozzi L, Giuntini C. Prevalence of airways obstruction in a general population: European Respiratory Society vs American Thoracic Society definition. Chest 2000;117(5 Suppl 2):339S–345S.
27. Connors AF Jr, Dawson NV, Thomas C, Harrell FE Jr, Desbiens N, Fulkerson WJ, et al. Outcomes following acute exacerbation of severe chronic obstructive lung disease. The SUPPORT investigators (Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments). Am J Respir Crit Care Med 1996;154(4 Pt 1):959–967.
28. Ferris BGJ, Anderson DO. The prevalence of chronic respiratory disease in a New Hampshire town. Am Rev Respir Dis 1962;86: 165–177.
29. Higgins MW, Keller JB, Landis JR, Beaty TH, Burrows B, Demets D, et al. Risk of chronic obstructive pulmonary disease: collaborative assessment of the validity of the Tecumseh index of risk. Am Rev Respir Dis 1984;130(3):380–385.
30. Mueller RE, Keble DL, Plummer J, Walker SH. The prevalence of chronic bronchitis, chronic airway obstruction, and respiratory symptoms in a Colorado city. Am Rev Respir Dis 1971;103(2):209–228.
31. Hurd SS. International efforts directed at attacking the problem of COPD. Chest 2000;117(5 Suppl 2):336S–338S.
32. Mannino DM, Gagnon RC, Petty TL, Lydick E. Obstructive lung disease and low lung function in adults in the United States: data from the National Health and Nutrition Examination Survey, 1988–1994. Arch Intern Med 2000;160(11):1683–1689.
33. Coultas DB, Mapel D, Gagnon R, Lydick E. The health impact of undiagnosed airflow obstruction in a national sample of United States adults. Am J Respir Crit Care Med 2001;164(3):372–377.
34. Pena VS, Miravitlles M, Gabriel R, Jimenez-Ruiz CA, Villasante C, Masa JF, et al. Geographic variations in prevalence and underdiagnosis of COPD: results of the IBERPOC multicentre epidemiological study. Chest 2000;118(4):981–989.
35. Surgeon General of the United States. The health consequences of smoking: chronic obstructive lung disease. Washington DC: Department of Health and Human Services Publication No. 84–50205, 1984.
36. Fletcher C, Peto R, Tinker C, et al. The natural history of chronic bronchitis and emphysema: an 8-year study of early chronic obstructive lung disease in working men in London. Oxford, New York: Oxford University Press, 1976.
37. Stang P, Lydick E, Silberman C, Kempel A, Keating ET. The prevalence of COPD: using smoking rates to estimate disease frequency in the general population. Chest 2000;117(5 Suppl 2):354S–359S.
38. Zielinski J, Bednarek M. Early detection of COPD in a high-risk population using spirometric screening. Chest 2001;119(3):731–736.
39. Hyatt RE. The interrelationship of pressure, flow, and volume during various respiratory maneuvers in normal and emphysematous patients. Am Rev Respir Dis 1961;83:676–683.
40. O'Donnell DE, Revill SM, Webb KA. Dynamic hyperinflation and exercise intolerance in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2001;164(5):770–777.
41. Hyatt RE. Expiratory flow limitation. J Appl Physiol 1983;55(1 Pt 1):1–7.
42. O'Donnell DE, Sanii R, Anthonisen NR, Younes M. Effect of dynamic airway compression on breathing pattern and respiratory sensation in severe chronic obstructive pulmonary disease. Am Rev Respir Dis 1987;135(4):912–918.
43. O'Donnell DE, Webb KA. Exertional breathlessness in patients with chronic airflow limitation: the role of lung hyperinflation. Am Rev Respir Dis 1993;148(5):1351–1357.
44. Camilli AE, Burrows B, Knudson RJ, Lyle SK, Lebowitz MD. Longitudinal changes in forced expiratory volume in one second in adults. Effects of smoking and smoking cessation. Am Rev Respir Dis 1987;135(4):794–799.
45. Walter R, Gottlieb DJ, O'Connor GT. Environmental and genetic risk factors and gene-environment interactions in the pathogenesis of chronic obstructive lung disease. Environ Health Perspect 2000; 108 Suppl 4:733–742.
46. Hale KA, Ewing SL, Gosnell BA, Niewoehner DE. Lung disease in long-term cigarette smokers with and without chronic air-flow obstruction. Am Rev Respir Dis 1984;130(5):716–721.
47. Takizawa H, Tanaka M, Takami K, Ohtoshi T, Ito K, Satoh M, et al. Increased expression of transforming growth factor- β_1 in small airway epithelium from tobacco smokers and patients with chronic obstructive pulmonary disease (COPD). Am J Respir Crit Care Med 2001;163(6):1476–1483.
48. Barnes PJ. Chronic obstructive pulmonary disease. N Engl J Med 2000;343(4):269–280.
49. Hill AT, Bayley D, Stockley RA. The interrelationship of sputum inflammatory markers in patients with chronic bronchitis. Am J Respir Crit Care Med 1999;160(3):893–898.
50. Keatings VM, Collins PD, Scott DM, Barnes PJ. Differences in interleukin-8 and tumor necrosis factor-α in induced sputum from patients with chronic obstructive pulmonary disease or asthma. Am J Respir Crit Care Med 1996;153(2):530–534.
51. Di Stefano A, Capelli A, Lusuardi M, Balbo P, Vecchio C, Maestrelli P, et al. Severity of airflow limitation is associated with severity of airway inflammation in smokers. Am J Respir Crit Care Med 1998;158(4):1277–1285.
52. Thompson AB, Daughton D, Robbins RA, Ghafouri MA, Oehlerking M, Rennard SI. Intraluminal airway inflammation in chronic bronchitis: characterization and correlation with clinical parameters. Am Rev Respir Dis 1989;140(6):1527–1537.
53. Retamales I, Elliott WM, Meshi B, Coxson HO, Pare PD, Sciurba FC, et al. Amplification of inflammation in emphysema and its association with latent adenoviral infection. Am J Respir Crit Care Med 2001;164(3):469–473.
54. Turato G, Di Stefano A, Maestrelli P, Mapp CE, Ruggieri MP, Roggeri A, et al. Effect of smoking cessation on airway inflammation in chronic bronchitis. Am J Respir Crit Care Med 1995;152(4 Pt 1):1262–1267.
55. Rutgers SR, Postma DS, ten Hacken NH, Kauffman HF, van Der Mark TW, Koeter GH, Timens W. Ongoing airway inflammation in

patients with COPD who do not currently smoke. Thorax 2000; 55(1):12–18.
56. Snider GL, Ciccolella DE, Morris SM, Stone PJ, Lucey EC. Putative role of neutrophil elastase in the pathogenesis of emphysema. Ann N Y Acad Sci 1991;624:45–59.
57. Hunninghake GW, Crystal RG. Cigarette smoking and lung destruction: accumulation of neutrophils in the lungs of cigarette smokers. Am Rev Respir Dis 1983;128(5):833–838.
58. Shapiro SD. Evolving concepts in the pathogenesis of chronic obstructive pulmonary disease. Clin Chest Med 2000;21(4):621–632.
59. Kanazawa H, Kurihara N, Hirata K, Fujimoto S, Takeda T. The role of free radicals and neutrophil elastase in development of pulmonary emphysema. Intern Med 1992;31(7):857–860.
60. Hautamaki RD, Kobayashi DK, Senior RM, Shapiro SD. Requirement for macrophage elastase for cigarette smoke-induced emphysema in mice. Science 1997;277(5334):2002–2004.
61. Snider GL. Clinical relevance summary: collagen vs elastin in pathogenesis of emphysema; cellular origin of elastases; bronchiolitis vs emphysema as a cause of airflow obstruction. Chest 2000;117(5 Suppl 1):244S–246S.
62. Saetta M, Di Stefano A, Turato G, Facchini FM, Corbino L, Mapp CE, et al. CD8+ T-lymphocytes in peripheral airways of smokers with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;157(3 Pt 1):822–826.
63. Finkelstein R, Fraser RS, Ghezzo H, Cosio MG. Alveolar inflammation and its relation to emphysema in smokers. Am J Respir Crit Care Med 1995;152(5 Pt 1):1666–1672.
64. Shapiro SD. The macrophage in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1999;160(5 Pt 2):S29–S32.
65. Finlay GA, O'Driscoll LR, Russell KJ, D'Arcy EM, Masterson JB, FitzGerald MX, O'Connor CM. Matrix metalloproteinase expression and production by alveolar macrophages in emphysema. Am J Respir Crit Care Med 1997;156(1):240–247.
66. Jeffery PK. Comparison of the structural and inflammatory features of COPD and asthma. Giles F. Filley Lecture. Chest 2000;117(5 Suppl 1):251S–260S.
67. Saetta M, Di Stefano A, Maestrelli P, Ferraresso A, Drigo R, Potena A, et al. Activated T-lymphocytes and macrophages in bronchial mucosa of subjects with chronic bronchitis. Am Rev Respir Dis 1993;147(2):301–306.
68. Saetta M, Baraldo S, Corbino L, Turato G, Braccioni F, Rea F, et al. CD8+ve cells in the lungs of smokers with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1999;160(2):711–717.
69. Diaz PT, King MA, Pacht ER, Wewers MD, Gadek JE, Nagaraja HN, et al. Increased susceptibility to pulmonary emphysema among HIV-seropositive smokers. Ann Intern Med 2000;132(5):369–372.
70. Diaz PT, Clanton TL, Pacht ER. Emphysema-like pulmonary disease associated with human immunodeficiency virus infection. Ann Intern Med 1992;116(2):124–128.
71. Diaz PT, King ER, Wewers MD, Gadek JE, Neal D, Drake J, Clanton TL. HIV infection increases susceptibility to smoking-induced emphysema. Chest 2000;117(5 Pt 1):285S.
72. Liu AN, Mohammed AZ, Rice WR, Fiedeldey DT, Liebermann JS, Whitsett JA, et al. Perforin-independent CD8+ T-cell-mediated cytotoxicity of alveolar epithelial cells is preferentially mediated by tumor necrosis factor-α: relative insensitivity to Fas ligand. Am J Respir Cell Mol Biol 1999;20(5):849–858.
73. Senior RM. Mechanisms of COPD: conference summary. Chest 2000;117(5 Suppl 1):320S–323S.
74. Driscoll KE, Carter JM, Hassenbein DG, Howard B. Cytokines and particle-induced inflammatory cell recruitment. Environ Health Perspect 1997;105 Suppl 5:1159–1164.
75. Devalia JL, Bayram H, Rusznak C, Calderon M, Sapsford RJ, Abdelaziz MA, et al. Mechanisms of pollution-induced airway disease: in vitro studies in the upper and lower airways. Allergy 1997; 52(38 Suppl):45–51;discussion 57–58.
76. Barnes PJ. Mechanisms in COPD: differences from asthma. Chest 2000;117(2 Suppl):10S–14S.
77. Shapiro SD, Kobayashi DK, Ley TJ. Cloning and characterization of a unique elastolytic metalloproteinase produced by human alveolar macrophages. J Biol Chem 1993;268(32):23824–23829.
78. Zheng T, Zhu Z, Wang Z, Homer RJ, Ma B, Riese RJ Jr, et al. Inducible targeting of IL-13 to the adult lung causes matrix metalloproteinase- and cathepsin-dependent emphysema. J Clin Invest 2000;106(9):1081–1093.
79. Wang Z, Zheng T, Zhu Z, Homer RJ, Riese RJ, Chapman HA Jr, et al. Interferon gamma induction of pulmonary emphysema in the adult murine lung. J Exp Med 2000;192(11):1587–1600.
80. Finlay GA, Russell KJ, McMahon KJ, D'arcy EM, Masterson JB, FitzGerald MX, O'Connor CM. Elevated levels of matrix metalloproteinases in bronchoalveolar lavage fluid of emphysematous patients. Thorax 1997;52(6):502–506.
81. Thomas PS, Yates DH, Barnes PJ. Tumor necrosis factor-α increases airway responsiveness and sputum neutrophilia in normal human subjects. Am J Respir Crit Care Med 1995;152(1):76–80.
82. Chamba A, Afford SC, Stockley RA, Burnett D. Extracellular proteolysis of fibronectin by neutrophils: characterization and the effects of recombinant cytokines. Am J Respir Cell Mol Biol 1991; 4(4):330–337.
83. Repine JE, Bast A, Lankhorst I. Oxidative stress in chronic obstructive pulmonary disease. Oxidative Stress Study Group. Am J Respir Crit Care Med 1997;156 (2 Pt 1)341–357.
84. Pratico D, Basili S, Vieri M, Cordova C, Violi F, Fitzgerald GA. Chronic obstructive pulmonary disease is associated with an increase in urinary levels of isoprostane $F_{2\alpha}$-III, an index of oxidant stress. Am J Respir Crit Care Med 1998;158(6):1709–1714.
85. Dekhuijzen PN, Aben KK, Dekker I, Aarts LP, Wielders PL, van Herwaarden CL, Bast A. Increased exhalation of hydrogen peroxide in patients with stable and unstable chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1996;154(3 Pt 1):813–816.
86. Tuder RM, Kasahara Y, Voelkel NF. Inhibition of vascular endothelial growth factor receptors causes emphysema in rats. Chest 2000;117(5 Suppl 1):281S.
87. Aoshiba K, Yasui S, Nagai A. Apoptosis of alveolar macrophages by cigarette smoke. Chest 2000;117(5 Suppl 1):320S.
88. Kasahara Y, Tuder RM, Cool CD, Lynch DA, Flores SC, Voelkel NF. Endothelial cell death and decreased expression of vascular endothelial growth factor and vascular endothelial growth factor receptor 2 in emphysema. Am J Respir Crit Care Med 2001;163(3 Pt 1):737–744.
89. Sethi S, Murphy TF. Bacterial infection in chronic obstructive pulmonary disease in 2000: a state-of-the-art review. Clin Microbiol Rev 2001;14(2):336–363.
90. Sethi S. Bacterial infection and the pathogenesis of COPD. Chest 2000;117(5 Suppl 1):286S–291S.
91. Barker DJ, Godfrey KM, Fall C, Osmond C, Winter PD, Shaheen SO. Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. BMJ 1991;303(6804):671–675.
92. Shaheen SO, Barker DJ, Shiell AW, Crocker FJ, Wield GA, Holgate ST. The relationship between pneumonia in early childhood and impaired lung function in late adult life. Am J Respir Crit Care Med 1994;149(3 Pt 1):616–619.
93. Johnston ID. Effect of pneumonia in childhood on adult lung function. J Pediatr 1999;135(2 Pt 2):33–37.

94. Shaheen SO, Sterne JA, Tucker JS, Florey CD. Birth weight, childhood lower respiratory tract infection, and adult lung function. Thorax 1998;53(7):549–553.
95. Keicho N, Elliott WM, Hogg JC, Hayashi S. Adenovirus E1A upregulates interleukin-8 expression induced by endotoxin in pulmonary epithelial cells. Am J Physiol 1997;272(6 Pt 1):L1046–L1052.
96. Keicho N, Elliott WM, Hogg JC, Hayashi S. Adenovirus E1A gene dysregulates ICAM-1 expression in transformed pulmonary epithelial cells. Am J Respir Cell Mol Biol 1997;16(1):23–30.
97. Matsumori A, Matoba Y, Sasayama S. Dilated cardiomyopathy associated with hepatitis C virus infection. Circulation 1995;92(9): 2519–2525.
98. Pouteil-Noble C, Maiza H, Dijoud F, MacGregor B. Glomerular disease associated with hepatitis C virus infection in native kidneys. Nephrol Dial Transplant 2000;15 Suppl 8:28–33.
99. Kubo K, Yamaguchi S, Fujimoto K, Hanaoka M, Hayasaka M, Honda T, et al. Bronchoalveolar lavage fluid findings in patients with chronic hepatitis C virus infection. Thorax 1996;51(3):312–314.
100. Anthonisen NR, Connett JE, Kiley JP, Altose MD, Bailey WC, Buist AS, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV_1. The Lung Health Study. JAMA 1994;272(19):1497–1505.
101. Tashkin DP, Altose MD, Connett JE, Kanner RE, Lee WW, Wise RA. Methacholine reactivity predicts changes in lung function over time in smokers with early chronic obstructive pulmonary disease. The Lung Health Study Research Group. Am J Respir Crit Care Med 1996;153(6 Pt 1):1802–1811.
102. Vestbo J, Prescott E, Lange P. Association of chronic mucus hypersecretion with FEV_1 decline and chronic obstructive pulmonary disease morbidity. Copenhagen City Heart Study Group. Am J Respir Crit Care Med 1996;153(5):1530–1535.
103. Prescott E, Lange P, Vestbo J. Chronic mucus hypersecretion in COPD and death from pulmonary infection. Eur Respir J 1995; 8(8):1333–1338.
104. Bowen JB, Votto JJ, Thrall RS, Haggerty MC, Stockdale-Woolley R, Bandyopadhyay T, ZuWallack RL. Functional status and survival following pulmonary rehabilitation. Chest 2000;118(3):697–703.
105. Keatings VM, Cave SJ, Henry MJ, Morgan K, O'Connor CM, FitzGerald MX, Kalsheker N. A polymorphism in the tumor necrosis factor-α gene promoter region may predispose to a poor prognosis in COPD. Chest 2000;118(4):971–975.
106. Dahl M, Tybjaerg-Hansen A, Vestbo J, Lange P, Nordestgaard BG. Elevated plasma fibrinogen associated with reduced pulmonary function and increased risk of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2001;164(6):1008–1011.
107. Buist AS, Van Fleet DL, Ross BB. A comparison of conventional spirometric tests and the test of closing volume in one emphysema screening center. Am Rev Respir Dis 1973;107(5):735–743.
108. Hodgkin JE. Prognosis in chronic obstructive pulmonary disease. Clin Chest Med 1990;11(3):555–569.
109. Schols AM. Nutrition in chronic obstructive pulmonary disease. Curr Opin Pulm Med 2000;6(2):110–115.
110. Schols AMWJ, Slangen J, Volovics L, Wouters EFM. Weight loss is a reversible factor in the prognosis of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;157(6 Pt 1): 1791–1797.
111. Bernard S, LeBlanc P, Whittom F, Carrier G, Jobin J, Belleau R, Maltais F. Peripheral muscle weakness in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998; 158(2):629–634.
112. Casaburi R. Skeletal muscle dysfunction in chronic obstructive pulmonary disease. Med Sci Sports Exerc 2001;33(7 Suppl):S662–S670.
113. Burrows B. Course and prognosis of patients with chronic airways obstruction. Chest 1980;77(2 Suppl):250–251.
114. Seneff MG, Wagner DP, Wagner RP, Zimmerman JE, Knaus WA. Hospital and 1-year survival of patients admitted to intensive care units with acute exacerbation of chronic obstructive pulmonary disease. JAMA 1995;274(23):1852–1857.
115. Brown PJ, Greville HW, Finucane KE. Asthma and irreversible airflow obstruction. Thorax 1984;39(2):131–136.
116. Prescott E, Lange P, Vestbo J. Socioeconomic status, lung function and admission to hospital for COPD: results from the Copenhagen City Heart Study. Eur Respir J 1999;13(5):1109–1114.
117. Poller W, Faber JP, Scholz S, Weidinger S, Bartholome K, Olek K, Eriksson S. Mis-sense mutation of α_1-antichymotrypsin gene associated with chronic lung disease (letter). Lancet 1992;339(8808): 1538.
118. Poller W, Faber JP, Weidinger S, Tief K, Scholz S, Fischer M, et al. A leucine-to-proline substitution causes a defective α_1- antichymotrypsin allele associated with familial obstructive lung disease. Genomics 1993;17(3):740–743.
119. Silverman EK, Speizer FE, Weiss ST, Chapman HA Jr, Schuette A, Campbell EJ, et al. Familial aggregation of severe, early-onset COPD: candidate gene approaches. Chest 2000;117(5 Suppl 1): 273S–274S.
120. Burchfiel CM, Enright PL, Sharp DS, Chyou PH, Rodriguez BL, Curb JD. Factors associated with variations in pulmonary function among elderly Japanese-American men. Chest 1997;112(1):87–97.
121. Pauwels RA, Löfdahl C-G, Laitinen LA, Schouten JP, Postma DS, Pride NB, Ohlsson SV. Long-term treatment with inhaled budesonide in persons with mild chronic obstructive pulmonary disease who continue smoking. European Respiratory Society Study on Chronic Obstructive Pulmonary Disease. N Engl J Med 1999; 340(25):1948–1953.
122. Sherman CB, Xu X, Speizer FE, Ferris BG Jr, Weiss ST, Dockery DW. Longitudinal lung function decline in subjects with respiratory symptoms. Am Rev Respir Dis 1992;146(4):855–859.
123. Mannino DM, Etzel RA, Flanders WD. Do the medical history and physical examination predict low lung function? Arch Intern Med 1993;153(16):1892–1897.
124. Badgett RG, Tanaka DJ, Hunt DK, Jelley MJ, Feinberg LE, Steiner JF, Petty TL. Can moderate chronic obstructive pulmonary disease be diagnosed by historical and physical findings alone? Am J Med 1993;94(2):188–196.
125. Mapel DW, Picchi MA, Hurley JS, Frost FJ, Petersen HV, Mapel VM, Coultas DB. Utilization in COPD: patient characteristics and diagnostic evaluation. Chest 2000;117(5 Suppl 2):346S–353S.
126. Chapman KR, Tashkin DP, Pye DJ. Gender bias in the diagnosis of COPD. Chest 2001;119(6):1691–1695.
127. van den Boom G, van Schayck CP, Rutten-van Mölken MPMH, Tirimanna PRS, den Otter JJ, van Grunsven PM, et al. Active detection of chronic obstructive pulmonary disease and asthma in the general population: results and economic consequences of the DIMCA program. Am J Respir Crit Care Med 1998;158(6):1730–1738.
128. National Lung Health Educational Program (NLHEP). Strategies in preserving lung health and preventing COPD and associated diseases. Chest 1998;113(2 Suppl):123S–163S.
129. Petty TL, Weinmann GG. Building a national strategy for the prevention and management of and research in chronic obstructive pulmonary disease. National Heart, Lung, and Blood Institute Workshop Summary. Bethesda, Maryland, August 29 -31, 1995. JAMA 1997;277(3):246–253.

130. Ferguson GT, Enright PL, Buist AS, Higgins MW. Office spirometry for lung health assessment in adults: a consensus statement from the National Lung Health Education Program. Respir Care 2000;45(5):513–530.
131. Hole DJ, Watt GCM, Davey-Smith G, Hart CL, Gillis CR, Hawthorne VM. Impaired lung functin and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. BMJ 1996;313(7059):711–715; discussion 715–716.
132. Wedzicha JA, Seemungal TA, MacCallum PK, Paul EA, Donaldson GC, Bhowmik A, et al. Acute exacerbations of chronic obstructive pulmonary disease are accompanied by elevations of plasma fibrinogen and serum IL-6 levels. Thromb Haemost 2000;84(2): 210–215.
133. Jones PW, Bosh TK. Quality of life changes in COPD patients treated with salmeterol. Am J Respir Crit Care Med 1997;155(4): 1283–1289.
134. Redelmeier DA, Bayoumi AM, Goldstein RS, Guyatt GH. Interpreting small differences in functional status: the Six Minute Walk test in chronic lung disease patients. Am J Respir Crit Care Med 1997;155(4):1278–1282.
135. Testa MA, Simonson DC. Assesment of quality-of-life outcomes. N Engl J Med 1996;334(13):835–840.
136. Guyatt GH, Feeny DH, Patrick DL. Measuring health-related quality of life. Ann Intern Med 1993;118(8):622–629.
137. Mahler DA, Jones PW. Measurement of dyspnea and quality of life in advanced lung disease. Clin Chest Med 1997;18(3):457–469.
138. Mahler DA. How should health-related quality of life be assessed in patients with COPD? Chest 2000;117(2 Suppl):54S–57S.
139. Rennard SI, Serby CW, Ghafouri M, Johnson PA, Friedman M. Extended therapy with ipratropium is associated with improved lung function in patients with COPD: a retrospective analysis of data from 7 clinical trials. Chest 1996;110(1):62–70.
140. Calverley P, Bellamy D. The challenge of providing better care for patients with chronic obstructive pulmonary disease: the poor relation of airways obstruction? Thorax 2000;55(1):78–82.
141. Barnes PJ. β-adrenergic receptors and their regulation. Am J Respir Crit Care Med 1995;152(3):838–860.
142. Johnson M, Rennard S. Alternative mechanisms for long-acting β_2-adrenergic agonists in COPD. Chest 2001;120(1):258–270.
143. Cook D, Guyatt G, Wong E, Goldstein R, Bedard M, Austin P, et al. Regular versus as-needed short-acting inhaled β-agonist therapy for chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2001;163(1):85–90.
144. Mahler DA, Donohue JF, Barbee RA, Goldman MD, Gross NJ, Wisniewski ME, et al. Efficacy of salmeterol xinafoate in the treatment of COPD. Chest 1999;115(4):957–965.
145. Dowling RB, Rayner CF, Rutman A, Jackson AD, Kanthakumar K, Dewar A, et al. Effect of salmeterol on *Pseudomonas aeruginosa* infection of respiratory mucosa. Am J Respir Crit Care Med 1997; 155(1):327–336.
146. Rhodes DG, Newton R, Butler R, Herbette L. Equilibrium and kinetic studies of the interactions of salmeterol with membrane bilayers. Mol Pharmacol 1992;42(4):596–602.
147. Johnson M. Salmeterol: a novel drug for the treatment of asthma. Agents Actions Suppl 1991;34:79–95.
148. Cazzola M, Di Perna F, Noschese P, Vinciguerra A, Calderaro F, Girbino G, Matera MG. Effects of formoterol, salmeterol or oxitropium bromide on airway responses to salbutamol in COPD. Eur Respir J 1998;11(6):1337–1341.
149. Celik G, Kayacan O, Beder S, Durmaz G. Formoterol and salmeterol in partially reversible chronic obstructive pulmonary disease: a crossover, placebo-controlled comparison of onset and duration of action. Respiration 1999;66(5):434–439.
150. Waldeck B. Some pharmacodynamic aspects on long-acting β-adrenoceptor agonists. Gen Pharmacol 1996;27(4):575–580.
151. Vervloet D, Ekstrom T, Pela R, Duce Gracia F, Kopp C, Silvert BD, et al. A 6-month comparison between formoterol and salmeterol in patients with reversible obstructive airways disease. Respir Med 1998;92(6):836–842.
152. Dahl R, Greefhorst LA, Nowak D, Nonikov V, Byrne AM, Thomson MH, et al. Inhaled formoterol dry powder versus ipratropium bromide in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2001;164(5):778–784.
153. Cazzola M, Matera MG, Santangelo G, Vinciguerra A, Rossi F, D'Amato G. Salmeterol and formoterol in partially reversible severe chronic obstructive pulmonary disease: a dose-response study. Respir Med 1995;89(5):357–362.
154. D'Urzo AD, De Salvo MC, Ramirez-Rivera A, Almeida J, Sichletidis L, Rapatz G, Kottakis J; FOR-INT-03 Study Group. In patients with COPD, treatment with a combination of formoterol and ipratropium is more effective than a combination of salbutamol and ipratropium: a 3-week, randomized, double-blind, within-patient, multicenter study. Chest 2001;119(5):1347–1356.
155. Cazzola M, Donner CF. Long-acting β_2 agonists in the management of stable chronic obstructive pulmonary disease. Drugs 2000; 60(2):307–320.
156. Ferguson GT, Cherniack RM. Management of chronic obstructive pulmonary disease. N Engl J Med 1993;328(14):1017–1022.
157. Karpel JP, Appel D, Breidbart D, Fusco MJ. A comparison of atropine sulfate and metaproterol sulfate in the emergency treatment of asthma. Am Rev Respir Dis 1986;133(5):727–729.
158. Martin RJ, Bartelson BL, Smith P, Hudgel DW, Lewis D, Pohl G, et al. Effect of ipratropium bromide treatment on oxygen saturation and sleep quality in COPD. Chest 1999;115(5):1338–1345.
159. Friedman M, Serby CW, Menjoge SS, Wilson JD, Hilleman DE, Witek TJ Jr. Pharmacoeconomic evaluation of a combination of ipratropium plus albuterol compared with ipratropium alone and albuterol alone in COPD. Chest 1999;115(3):635–641.
160. COMBIVENT Inhalation Aerosol Study Group. In chronic obstructive pulmonary disease, a combination of ipratropium bromide and albuterol is more effective than either agent alone. An 85-day multicenter trial. Chest 1994;105(5):1411–1419.
161. Dorinsky PM, Reisner C, Ferguson GT, Menjoge SS, Serby CW, Witek TJ Jr. The combination of ipratropium and albuterol optimizes pulmonary function reversibility testing in patients with COPD. Chest 1999;115(4):966–971.
162. Sin DD, Tu JV. Lack of association between ipratropium bromide and mortality in elderly patients with chronic obstructive airway disease. Thorax 2000;55(3):194–197.
163. Rennard SI, Anderson W, ZuWallack R, Broughton J, Bailey W, Friedman M, et al. Use of a long-acting inhaled β_2-adrenergic agonist, salmeterol xinafoate, in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2001;163(5):1087–1092.
164. van Noord JA, Bantje TA, Eland ME, Korducki L, Cornelissen PJ. A randomised controlled comparison of tiotropium nd ipratropium in the treatment of chronic obstructive pulmonary disease. The Dutch Tiotropium Study Group. Thorax 2000;55(4):289–294.
165. Casaburi R, Briggs DD Jr, Donohue JF, Serby CW, Menjoge SS, Witek TJ Jr. The spirometric efficacy of once-daily dosing with tiotropium in stable COPD: a 13-week multicenter trial. The US Tiotropium Study Group. Chest 2000;118(5):1294–1302.
166. Littner MR, Howite JS, Tashkin DP, Friedman M, Serby CW, Menjoge SS, Witek TJ Jr. Long-acting bronchodilation with once-daily dosing of tiotropium (Spiriva) in stable chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2000;161(4 Pt 1):1136–1142.

167. Vaz Fragoso CA, Muller MA. Review of the clinical efficacy of theophylline in the treatment of chronic obstructive pulmonary disease. Am Rev Respir Dis 1993;147(6 Pt 2):S40–S47.
168. McKay SE, Howie CA, Thompson AH, Whiting B, Addis GJ. Value of theophylline treatment in patients handicapped by chronic obstructive lung disease. Thorax 1993;48(3):227–232.
169. ZuWallack RL, Mahler DA, Reilly D, Church N, Emmett A, Rickard K, Knobil K. Salmeterol plus theophylline combination therapy in the treatment of COPD. Chest 2001;119(6):1661–1670.
170. Keatings VM, Jatakanon A, Worsdell YM, Barnes PJ. Effects of inhaled and oral glucocorticoids on inflammatory indices in asthma and COPD. Am J Respir Crit Care Med 1997;155(2):542–548.
171. Culpitt SV, Maziak W, Loukidis S, Nightingale JA, Matthews JL, Barnes PJ. Effect of high dose inhaled steroid on cells, cytokines, and proteases in induced sputum in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1999;160(5 Pt 1):1635–1639.
172. Yildiz F, Kaur AC, Ilgazli A, Celikoglu M, Kacar Ozkara S, Paksoy N, Ozkarakas O. Inhaled corticosteroids may reduce neutrophilic inflammation in patients with stable chronic obstructive pulmonary disease. Respiration 2000;67(1):71–76.
173. Meagher LC, Cousin JM, Seckl JR, Haslett C. Opposing effects of glucocorticoids on the rate of apoptosis in neutrophilic and eosinophilic granulocytes. J Immunol 1996;156(11):4422–4428.
174. Decramer M, de Bock V, Dom R. Functional and histologic picture of steroid-induced myopathy in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1996;153(6 Pt 1):1958–1964.
175. Rice KL, Rubins JB, Lebahn F, Parenti CM, Duane PG, Kuskowski M, et al. Withdrawal of chronic systemic corticosteroids in patients with COPD: a randomized trial. Am J Respir Crit Care Med 2000; 162(1):174–178.
176. Decramer M, Lacquet LM, Fagard R, Rogiers P. Corticosteroids contribute to muscle weakness in chronic airflow obstruction. Am J Respir Crit Care Med 1994;150(1):11–16.
177. Decramer M, Stas KJ. Corticosteroid-induced myopathy involving respiratory muscles in patients with chronic obstructive pulmonary disease or asthma. Am Rev Respir Dis 1992;146(3):800–802.
178. Burge PS, Calverley PM, Jones PW, Spencer S, Anderson JA, Maslen TK. Randomised, double blind, placebo controlled study of fluticasone propionate in patients with moderate to severe chronic obstructive pulmonary disease: the ISOLDE trial. BMJ 2000; 320(7245):1297–1303.
179. Senderovitz T, Vestbo J, Frandsen J, Maltbaek N, Norgaard M, Nielsen C, Kampmann JP. Steroid reversibility test followed by inhaled budesonide or placebo in outpatients with stable chronic obstructive pulmonary disease. The Danish Society of Respiratory Medicine. Respir Med 1999;93(10):715–718.
180. Ferreira IM, Hazari MS, Gutierrez C, Zamel N, Chapman KR. Exhaled nitric oxide and hydrogen peroxide in patients with chronic obstructive pulmonary disease: effects of inhaled beclomethasone. Am J Respir Crit Care Med 2001;164(6):1012–1015.
181. Sin DD, Tu JV. Inhaled corticosteroids and the risk of mortality and readmission in elderly patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2001;164(4):580–584.
182. Effect of inhaled triamcinolone on the decline in pulmonary function in chronic obstructive pulmonary disease. N Engl J Med 2000; 343(26):1902–1909.
183. Paggiaro PL, Dahle R, Bakran I, Frith L, Hollingworth K, Efthimiou J. Multicentre randomised placebo-controlled trial of inhaled fluticasone propionate in patients with chronic obstructive pulmonary disease. International COPD Study Group. Lancet 1998; 351(9105):773–780. [erratum in Lancet 1998;351(9120):1968]
184. Vestbo J, Sorensen T, Lange P, Brix A, Torre P, Viskum K. Long-term effect of inhaled budesonide in mild and moderate chronic obstructive pulmonary disease: a randomised controlled trial. Lancet 1999;353(9167):1819–1823.
185. Spencer S, Calverley PM, Sherwood Burge P, Jones PW. Health status deterioration in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2001;163(1):122–128.
186. O'Brien A, Russo-Magno P, Karki A, Hiranniramol S, Hardin M, Kaszuba M, et al. Effects of withdrawal of inhaled steroids in men with severe irreversible airflow obstruction. Am J Respir Crit Care Med 2001;164(3):365–371.
187. Israel E, Banerjee TR, Fitzmaurice GM, Kotlov TV, LaHive K, LeBoff MS. Effects of inhaled glucocorticoids on bone density in premenopausal women. N Engl J Med 2001;345(13):941–947.
188. Hosenpud JD, Bennett LE, Keck BM, Edwards EB, Novick RJ. Effect of diagnosis on survival benefit of lung transplantation for end-stage lung disease. Lancet 1998;351(9095):24–27.
189. Hosenpud JD, Bennett LE, Keck BM, Fiol B, Boucek MM, Novick RJ. The Registry of the International Society for Heart and Lung Transplantation: sixteenth official report—1999. J Heart Lung Transplant 1999;18(7):611–626.
190. Maurer JR, Frost AE, Estenne M, Higenbottam T, Glanville AR. International guidelines for the selection of lung transplant candidates. The International Society for Heart and Lung Transplantation, the American Thoracic Society, the American Society of Transplant Physicians, the European Respiratory Society. Transplantation 1998;66(7):951–956.
191. Gartner SH, Sevick MA, Keenan RJ, Chen GJ. Cost-utility of lung transplantation: a pilot study. J Heart Lung Transplant 1997;16(11): 1129–1134.
192. Al MJ, Koopmanschap MA, van Enckevort PJ, Geertsma A, van der Bij W, de Boer WJ, TenVergert EM. Cost-effectiveness of lung transplantation in The Netherlands: a scenario analysis. Chest 1998; 113(1):124–130.
193. Division of Transportation BoHRD. Annual Report of the US Registry for Transplant Recipients and the Organ Procurement and Transplantation Network-Transplant Data: 1988–1994. Rockville, MD: US Dept of Health and Human Services, Health Resources and Services Administration, 1995.
194. Rationale and design of the National Emphysema Treatment Trial: a prospective randomized trial of lung volume reduction surgery. The National Emphysema Treatment Trial Research Group. Chest 1999;116(6):1750–1761.
195. Patients at high risk of death after lung-volume-reduction surgery. N Engl J Med 2001;345(15):1075–1083.
196. Naunheim KS, Kaiser LR, Bavaria JE, Hazelrigg SR, Magee MJ, Landreneau RJ, et al. Long-term survival after thoracoscopic lung volume reduction: a multiinstitutional review. Ann Thorac Surg 1999;68(6):2026–2031;discussion 2031–2032.
197. Flaherty KR, Kazerooni EA, Curtis JL, Iannettoni M, Lange L, Schork MA, Martinez FJ. Short-term and long-term outcomes after bilateral lung volume reduction surgery : prediction by quantitative CT. Chest 2001;119(5):1337–1346.
198. Hamacher J, Russi EW, Weder W, Lung volume reduction surgery: a survey on the European experience. Chest 2000;117(6):1560–1567.
199. Meyers BF, Yusen RD, Lefrak SS, Patterson GA, Pohl MS, Richardson VJ, Cooper JD. Outcome of Medicare patients with emphysema selected for, but denied, a lung volume reduction operation. Ann Thorac Surg 1998;66(2):331–336.
200. Gelb AF, McKenna RJ, Brenner M, Schein MJ, Zamel N, Fischel R. Lung function 4 years after lung volume reduction surgery for emphysema. Chest 1999;116(6):1608–1615.
201. Gelb AF, McKenna RJ Jr, Brenner M, Epstein JD, Zamel N. Lung function 5 yr after lung volume reduction surgery for emphysema. Am J Respir Crit Care Med 2001;163(7):1562–1566.

202. Geddes D, Davies M, Koyama H, Hansell D, Pastorino U, Pepper J, et al. Effect of lung-volume-reduction surgery in patients with severe emphysema. N Engl J Med 2000;343(4):239–245.
203. Christensen PJ, Paine R 3rd, Curtis JL, Kazerooni EA, Iannettoni MD, Martinez FJ. Weight gain after lung volume reduction surgery is not correlated with improvement in pulmonary mechanics. Chest 1999;116(6):1601–1607.
204. Martinez FJ, de Oca MM, Whyte RI, Stetz J, Gay SE, Celli BR. Lung-volume reduction improves dyspnea, dynamic hyperinflation, and respiratory muscle function. Am J Respir Crit Care Med 1997; 155(6):1984–1990.
205. Cassart M, Hamacher J, Verbandt Y, Wildermuth S, Ritscher D, Russi EW, et al. Effects of lung volume reduction surgery for emphysema on diaphragm dimensions and configuration. Am J Respir Crit Care Med 2001;163(5):1171–1175.
206. O'Donnell DE, Webb KA, Bertley JC, Chau LK, Conlan AA. Mechanisms of relief of exertional breathlessness following unilateral bullectomy and lung volume reduction surgery in emphysema. Chest 1996;110(1):18–27.
207. Benditt JO, Wood DE, McCool FD, Lewis S, Albert RK. Changes in breathing and ventilatory muscle recruitment patterns induced by lung volume reduction surgery. Am J Respir Crit Care Med 1997; 155(1):279–284.
208. Criner G, Cordova FC, Leyenson V, Roy B, Travaline J, Sudarshan S, et al. Effect of lung volume reduction surgery on diaphragm strength. Am J Respir Crit Care Med 1998;157(5 Pt 1):1578–1585.
209. Laghi F, Jubran A, Topeli A, Fahey PJ, Garrity ER Jr, Arcidi JM, et al. Effect of lung volume reduction surgery on neuromechanical coupling of the diaphragm. Am J Respir Crit Care Med 1998; 157(2):475–483.
210. Leyenson V, Furukawa S, Kuzma AM, Cordova F, Travaline J, Criner GJ. Correlation of changes in quality of life after lung volume reduction surgery with changes in lung function, exercise, and gas exchange. Chest 2000;118(3):728–735.
211. Rogers RM, Coxson HO, Sciurba FC, Keenan RJ, Whittall KP, Hogg JC. Preoperative severity of emphysema predictive of improvement after lung volume reduction surgery: use of CT morphometry. Chest 2000;118(5):1240–1247.
212. Gierada DS, Slone RM, Bae KT, Yusen RD, Lefrak SS, Cooper JD. Pulmonary emphysema: comparison of preoperative quantitative CT and physiologic index values with clinical outcome after lung-volume reduction surgery. Radiology 1997;205(1):235–242.
213. Elpern EH, Behner KG, Klontz B, Warren WH, Szidon JP, Kesten S. Lung volume reduction surgery: an analysis of hospital costs. Chest 1998;113(4):896–899.
214. Meyers BF, Patterson GA. Lung transplantation versus lung volume reduction as surgical therapy for emphysema. World J Surg 2001;25(2):238–243.
215. Meecham Jones DJ, Paul EA, Jones PW, Wedzicha JA. Nasal pressure support ventilation plus oxygen compared with oxygen therapy alone in hypercapnic COPD. Am J Respir Crit Care Med 1995; 152(2):538–544.
216. Casanova C, Celli BR, Tost L, Soriano E, Abreu J, Velasco V, Santolaria F. Long-term controlled trial of nocturnal nasal positive pressure ventilation in patients with severe COPD. Chest 2000; 118(6):1582–1590.
217. Lin CC. Comparison between nocturnal nasal positive pressure ventilation combined with oxygen therapy and oxygen montherapy in patients with severe COPD. Am J Respir Crit Care Med 1996; 154(2 Pt 1):353–358.
218. Strumpf DA, Millman RP, Carlisle CC, Gratton LM, Ryan SM, Erickson AD, Hill NS. Nocturnal positive-pressure ventilation via nasal mask in patients with severe chronic obstructive pulmonary disease. Am Rev Respir Dis 1991;144(6):1234–1239.
219. Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines. ACCP/AACVPR Pulmonary Rehabilitation Guidelines Panel. American College of Chest Physicians. American Association of Cardiovascular and Pulmonary Rehabilitation. Chest 1997; 112(5):1363–1396.
220. Lacasse Y, Wong E, Guyatt GH, King D, Cook DJ, Goldstein RS. Meta-analysis of respiratory rehabilitation in chronic obstructive pulmonary disease. Lancet 1996;348(9035):1115–1119.
221. Berry MJ, Rejeski WJ, Adair NE, Zaccaro D. Exercise rehabilitation and chronic obstructive pulmonary disease stage. Am J Respir Crit Care Med 1999;160(4):1248–1253.
222. Foglio K, Bianchi L, Bruletti G, Battista L, Pagani M, Ambrosino N. Long-term effectiveness of pulmonary rehabilitation in patients with chronic airway obstruction. Eur Respir J 1999;13(1):125–132.
223. Young P, Dewse M, Fergusson W, Kolbe J. Improvements in outcomes for chronic obstructive pulmonary disease (COPD) attributable to a hospital-based respiratory rehabilitation programme. Aust N Z J Med 1999;29(1):59–65.
224. Foglio K, Bianchi L, Ambrosino N. Is it really useful to repeat outpatient pulmonary rehabilitation programs in patients with chronic airway obstruction? A 2-year controlled study. Chest 2001; 119(6):1696–1704.
225. Finnerty JP, Keeping I, Bullough I, Jones J. The effectiveness of outpatient pulmonary rehabilitation in chronic lung disease: a randomized controlled trial. Chest 2001;119(6):1705–1710.
226. Kozora E, Make BJ. Cognitive improvement following rehabilitation in patients with COPD. Chest 2000;117():249S.
227. Heffner JE. Role of pulmonary rehabilitation in palliative care. Respir Care 2000;45(11):1365–1371;discussion 1371–1375.
228. Heffner JE. End-of-life issues. Respir Care Clin N Am 1998;4(3): 541–559.
229. Heffner JE. Chronic obstructive pulmonary disease: ethical considerations of care. Clin Pulm Med 1996;3:1–8.
230. Heffner JE, Fahy B, Hilling L, Barbieri C. Outcomes of advance directive education of pulmonary rehabilitation patients. Am J Respir Crit Care Med 1997;155(3):1055–1059.
231. Heffner JE, Fahy B, Hilling L, Barbieri C. Attitudes regarding advance directives among patients in pulmonary rehabilitation. Am J Respir Crit Care Med 1996;154(6 Pt 1):1735–1740.
232. Heffner JE, Fahy B, Barbieri C. Advance directive education during pulmonary rehabilitation. Chest 1996;109(2):373–379.
233. Seemungal TA, Donaldson GC, Paul EA, Bestall JC, Jeffries DJ, Wedzicha JA. Effect of exacerbation on quality of life in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;157(5 Pt 1):1418–1422.
234. Seemungal TA, Donaldson GC, Bhowmik A, Jeffries DJ, Wedzicha JA. Time course and recovery of exacerbations in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2000;161(5):1608–1613.
235. Snow V, Lascher S, Mottur-Pilson C. The evidence base for management of acute exacerbations of COPD: clinical practice guideline, part 1. Chest 2001;119(4):1185–1189.
236. Anthonisen NR, Manfreda J, Warren CP, Hershfield ES, Harding GK, Nelson NA. Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. Ann Intern Med 1987;106(2):196–204.
237. Bhowmik A, Seemungal TA, Sapsford RJ, Wedzicha JA. Relation of sputum inflammatory markers to symptoms and lung function changes in COPD exacerbations. Thorax 2000;55(2):114–120.
238. Saetta M, Di Stefano A, Maestrelli P, Turato G, Ruggieri MP, Roggeri A, et al. Airway eosinophilia in chronic bronchitis during exacerbations. Am J Respir Crit Care Med 1994;150(6 Pt 1):1646–1652.

239. Saetta M, Di Stefano A, Maestrelli P, Turato G, Mapp CE, Pieno M, et al. Airway eosinophilia and expression of interleukin-5 protein in asthma and in exacerbations of chronic bronchitis. Clin Exp Allergy 1996;26(7):766–774.
240. Zhu J, Qiu YS, Majumdar S, Gamble E, Matin D, Turato G, et al. Exacerbations of bronchitis: bronchial eosinophilia and gene expression for interleukin-4, interleukin-5, and eosinophil chemoattractants. Am J Respir Crit Care Med 2001;164(1):109–116.
241. Stockley RA, O'Brien C, Pye A, Hill SL. Relationship of sputum color to nature and outpatient management of acute exacerbations of COPD. Chest 2000;117(6):1638–1645.
242. Stockley RA. New approaches to the management of COPD. Chest 2000;117(2 Suppl):58S–62S.
243. Anderson HR, Spix C, Medina S, Schouten JP, Castellsague J, Rossi G, et al. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. Eur Respir J 1997;10(5):1064–1071.
244. Donaldson GC, Seemungal T, Jeffries DJ, Wedzicha JA. Effect of temperature on lung function and symptoms in chronic obstructive pulmonary disease. Eur Respir J 1999;13(4):844–849.
245. Chodosh S, McCarty J, Farkas S, Drehobl M, Tosiello R, Shan M, et al. Randomized, double-blind study of ciprofloxacin and cefuroxime axetil for treatment of acute bacterial exacerbations of chronic bronchitis. The Bronchitis Study Group. Clin Infect Dis 1998;27(4):722–729.
246. Logan WPD. Mortality in the London fog incident. Lancet 1953;1:336–338.
247. Thurston GD, Ito K. Epidemiological studies of ozone exposure effects. In: Holgate ST, Samet JM, Koren HS, et al, editors. Air pollution and health. London: Academic Press; 1999:485–510.
248. Pope CA 3rd, Kanner RE. Acute effects of PM_{10} pollution on pulmonary function of smokers with mild to moderate chronic obstructive pulmonary disease. Am Rev Respir Dis 1993;147(6 Pt 1):1336–1340.
249. Dockery DW, Pope CA. Epidemiology of acute health effects: summary of time series studies. In: Wilson R, Spangler J, editors. Particles in our air. Cambridge, MA: Harvard University Press; 1996:123–132.
250. Ghio AJ, Samet JM. Metals and air pollution particles. In: Hogate ST, Samet JM, editors. Air pollution and health. London: Academic Press; 1999:635–651.
251. MacNee W, Donaldson K. Exacerbations of COPD: environmental mechanisms. Chest 2000;117(5 Suppl 2):390S–397S.
252. Jany B, Gallup M, Tsuda T, Basbaum C. Mucin gene expression in rat airways following infection and irritation. Biochem Biophys Res Commun 1991;181(1):1–8.
253. Li XY, Gilmour PS, Donaldson K, MacNee W. Free radical activity and pro-inflammatory effects of particulate air pollution (PM_{10}) in vivo and in vitro. Thorax 1996;51(12):1216–1222.
254. Jimenez LA, Thompson J, Brown DA, Rahman I, Antonicelli F, Duffin R, et al. Activation of NF-κB by PM_{10} occurs via an iron-mediated mechanism in the absence of IκB degradation. Toxicol Appl Pharmacol 2000;166(2):101–110.
255. Read RC, Wilson R, Rutman A, Lund V, Todd HC, Brian AP, et al. Interaction of nontypable *Haemophilus influenzae* with human respiratory mucosa in vitro. J Infect Dis 1991;163(3):549–558.
256. Sethi S. Infectious etiology of acute exacerbations of chronic bronchitis. Chest 2000;117(5 Suppl 2):380S–385S.
257. Tager I, Speizer FE. Role of infection in chronic bronchitis. N Engl J Med 1975;292(11):563–571.
258. Soler N, Torres A, Ewig S, Gonzalez J, Ceis R, El-Ebiary M, et al. Bronchial microbial patterns in severe exacerbations of chronic obstructive pulmonary disease (COPD) requiring mechanical ventilation. Am J Respir Crit Care Med 1998;157(5 Pt 1):1498–1505.
259. Monso E, Ruiz J, Rosell A, Manterola J, Fiz J, Morera J, Ausina V. Bacterial infection in chronic obstructive pulmonary disease: a study of stable and exacerbated outpatients using the protected specimen brush. Am J Respir Crit Care Med 1995;152(4 Pt 1):1316–1320.
260. Fagon JY, Chastre J, Trouillet JL, Domart Y, Dombret MC, Bornet M, Gibert C. Characterization of distal bronchial microflora during acute exacerbation of chronic bronchitis: use of the protected specimen brush technique in 54 mechanically ventilated patients. Am Rev Respir Dis 1990;142(5):1004–1008.
261. Pela R, Marchesani F, Agostinelli C, Staccioli D, Cecarini L, Bassotti C, Sanguinetti CM. Airways microbial flora in COPD patients in stable clinical conditions and during exacerbations: a bronchoscopic investigation. Monaldi Arch Chest Dis 1998;53(3):262–267.
262. Murphy TF, Sethi S. Bacterial infection in chronic obstructive pulmonary disease. Am Rev Respir Dis 1992;146(4):1067–1083.
263. Yi K, Sethi S, Murphy TF. Human immune response to nontypeable *Haemophilus influenzae* in chronic bronchitis. J Infect Dis 1997;176(5):1247–1252.
264. Blasi F, Legnani D, Lombardo VM, Negretto GG, Magliano E, Pozzoli R, et al. *Chlamydia pneumoniae* infection in acute exacerbations of COPD. Eur Respir J 1993;6(1):19–22.
265. Gump DW, Phillips CA, Forsyth BR, McIntosh K, Lamborn KR, Stouch WH. Role of infection in chronic bronchitis. Am Rev Respir Dis 1976;113(4):465–474.
266. Matsuse T, Hayashi S, Kuwano K, Keunecke H, Jefferies WA, Hogg JC. Latent adenoviral infection in the pathogenesis of chronic airways obstruction. Am Rev Respir Dis 1992;146(4):177–184.
267. Weinberger M, Oddone EZ, Henderson WG. Does increased access to primary care reduce hospital readmissions? Veterans Affairs Cooperative Study Group on Primary Care and Hospital Readmission. N Engl J Med 1996;334(22):1441–1447.
268. Mushlin AI, Black ER, Connolly CA, Buonaccorso KM, Eberly SW. The necessary length of hospital stay for chronic pulmonary disease. JAMA 1991;266(1):80–83.
269. Connors AF Jr, McCaffree DR, Gray BA. Effect of inspiratory flow rate on gas exchange during mechanical ventilation. Am Rev Respir Dis 1981;124(5):537–543.
270. Garcia-Aymerich J, Monso E, Marrades RM, Escarrabill J, Felez MA, Sunyer J, Auto JM. Risk Factors for hospitalization for a chronic obstructive pulmonary disease exacerbation. EFRAM study. Am J Respir Crit Care Med 2001;164(6):1002–1007.
271. Niewoehner DE, Collins D, Erbland ML. Relation of FEV_1 to clinical outcomes during exacerbations of chronic obstructive pulmonary disease. Department of Veterans Affairs Cooperative Study Group. Am J Respir Crit Care Med 2000;161(4 Pt 1):1201–1205.
272. Nevins ML, Epstein SK. Predictors of outcome for patients with COPD requiring invasive mechanical ventilation. Chest 2001;119(6):1840–1849.
273. Snow V, Lascher S, Mottur-Pilson C. Evidence base for management of acute exacerbations of chronic obstructive pulmonary disease. Ann Intern Med 2001;134(7):595–599.
274. Murata GH, Gorby MS, Kapsner CO, Chick TW, Halperin AK. A multivariate model for the prediction of relapse after outpatient treatment of decompensated chronic obstructive pulmonary disease. Arch Intern Med 1992;152(1):73–77.
275. Evidence Report/Technology Assessment: Number 19. Management of Acute Exacerbations of Chronic Obstructive Pulmonary Disease: Summary. Available at http://www.ahrq.gov/clinic/epcsums/copdsum.htm (accessed 3/13/02).
276. Niewoehner DE. Role of corticosteroids in acute exacerbations of chronic obstructive pulmonary disease. Curr Opin Pulm Med 2001;7(2):75–78.
277. Niewoehner DE, Erbland ML, Deupree RH, Collins D, Gross NJ, Light RW, et al. Effect of systemic glucocorticoids on exacerba-

tions of chronic obstructive pulmonary disease. Department of Veterans Affairs Cooperative Study Group. N Engl J Med 1999;340(25): 1941–1947.
278. Angus RM, Ahmed AA, Fenwick LJ, Peacock AJ. Comparison of the acute effects on gas exchange of nasal ventilation and doxapram in exacerbations of chronic obstructive pulmonary disease. Thorax 1996;51(10):1048–1050.
279. Barbé F, Togores B, Rubi M, Pons S, Maimo A, Agusti AG. Noninvasive ventilatory support does not facilitate recovery from acute respiratory failure in chronic obstructive pulmonary disease. Eur Respir J 1996;9(6):1240–1245.
280. Bott J, Carroll MP, Conway JH, Keilty SE, Ward EM, Brown AM, et al. Randomised controlled trial of nasal ventilation in acute ventilatory failure due to chronic obstructive airways disease. Lancet 1993;341(8860):1555–1557.
281. Brochard L, Mancebo J, Wysocki M, Lofasso F, Conti G, Rauss A, et al. Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. N Engl J Med 1995;333(13):817–822.
282. Kramer N, Meyer TJ, Meharg J, Cece RD, Hill NS. Randomized, prospective trial of noninvasive positive pressure ventilation in acute respiratory failure. Am J Respir Crit Care Med 1995;151(6):1799–1806.
283. Plant PK, Owen JL, Elliott MW. One year period prevalence study of respiratory acidosis in acute exacerbations of COPD: implications for the provision of non-invasive ventilation and oxygen administration. Thorax 2000;55(7):550–554.
284. Plant PK, Owen JL, Elliott MW. Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial. Lancet 2000;355(9219):1931–1935.
285. Keenan SP. Noninvasive positive pressure ventilation in acute respiratory failure. Jama 2000;284(18):2376–2378.
286. Nava S, Ambrosino N, Clini E, Prato M, Orlando G, Vitacca M, et al. Noninvasive mechanical ventilation in the weaning of patients with respiratory failure due to chronic obstructive pulmonary disease: a randomized controlled trial. Ann Intern Med 1998;128(9): 721–728.
287. Hilbert G, Gruson D, Portel L, Gbikpi-Benissan G, Cardinaud JP. Noninvasive pressure support ventilation in COPD patients with postextubation hypercapnic respiratory insufficiency. Eur Respir J 1998;11(6):1349–1353.
288. International Consensus Conferences in Intensive Care Medicine: noninvasive positive pressure ventilation in acute respiratory failure. Am J Respir Crit Care Med 2001;163(1):283–291.
289. Doherty MJ, Greenstone MA. Survey of non-invasive ventilation (NIPPV) in patients with acute exacerbations of chronic obstructive pulmonary disease (COPD) in the UK. Thorax 1998;53(10):863–866.
290. Salin-Pascual RJ, Rosas M, Jimenez-Genchi A, Rivera-Meza BL, Delgado-Parra V. Antidepressant effect of transdermal nicotine patches in nonsmoking patients with major depression. J Clin Psychiatry 1996;57(9):387–389.
291. Hughes JR, Higgins ST, Bickel WK. Nicotine withdrawal versus other drug withdrawal syndromes: similarities and dissimilarities. Addiction 1994;89(11):1461–1470.
292. Carmelli D, Swan GE, Robinette D, Fabsitz R. Genetic influence on smoking: a study of male twins. N Engl J Med 1992;327(12):829–833.
293. Rossing MA. Genetic influences on smoking: candidate genes. Environ Health Perspect 1998;106(5):231–238.
294. Pianezza ML, Sellers EM, Tyndale RF. Nicotine metabolism defect reduces smoking. Nature 1998;393(6687):750.
295. Jorenby DE, Leischow SJ, Nides MA, Rennard SI, Johnston JA, Hughes AR, et al. A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. N Engl J Med 1999;340(9):685–691.
296. McNicholas WT, Levy P. Sleep-related breathing disorders: definitions and measurements. Eur Respir J 2000;15(6):988–989.
297. McNicholas WT. Impact of sleep in COPD. Chest 2000;117(2 Suppl):48S–53S.
298. Massaro D, Massaro GD. Pulmonary alveolus formation: critical period, retinoid regulation and plasticity. Novartis Found Symp 2001;234:229–236.
299. Belloni PN, Garvin L, Mao CP, Bailey-Healy I, Leaffer D. Effects of all-trans-retinoic acid in promoting alveolar repair. Chest 2000; 117(5 Suppl):235S–241S.
300. Gross CP, Anderson GF, Powe NR. The relation between funding by the National Institutes of Health and the burden of disease. N Engl J Med 1999;340(24):1881–1887.

Books, Films, Tapes, & Software

Managing Death in the Intensive Care Unit: The Transition From Cure to Comfort. J Randall Curtis and Gordon D Rubenfeld, Editors. New York: Oxford University Press. 2001. Hard cover, 388 pages, $59.95.

Managing Death in the Intensive Care Unit represents the definitive text on end-of-life care in the intensive care unit (ICU). This work is a refreshing attempt to redefine death in the ICU and will serve as a comprehensive resource for health care professionals caring for critically ill patients.

The text is organized into 5 parts. Part I describes death and the role of critical care clinicians in the ICU. Provocative ethical vignettes throughout this part serve to emotionally involve the reader as well. The next 2 parts are complementary: the transition from cure to supportive care is described in Part II, and then practical skills for making the transition are outlined in Part III. Part IV places all of the aforementioned issues in the larger societal context. Part V addresses aspects of the dying process germane to 7 specific patient populations.

Although the readership of Respiratory Care Journal is predominately respiratory therapists and physicians, **Managing Death in the Intensive Care Unit** has application across multiple disciplines, including all health care professionals who care for critically ill patients.

Concise chapters and a comprehensive index allow the reader to rapidly review the most recent literature on a particular topic. This organization facilitates using the book as a reference manual and resource for references from which to gather more specific information.

The chapter devoted to withdrawal of care is complete with sample order sheets and provides practical tools to clinicians who have anxiety about the process of withdrawing life-sustaining treatments. A practical addition would have been a similar chapter on advance directives. Although advance directives are mentioned in several sections, advance directives, living wills, and power of attorney in the critical care setting warrant additional discussion.

The only chapter that required substantial clarification was the one on legal liability. Reference is made to a number of court cases, but there is no explanation of the individual cases, making the reading somewhat cumbersome. Typographical errors were uncommon and when present of little importance.

The chapters are well referenced, with an appropriate balance of scientific and lay references. The one exception is the chapter entitled "Making a Personal Relationship with Death," where there are, surprisingly, no references. Though that may have been an oversight, it is also possible that there is a paucity of investigation on that subject.

Throughout the text the authors explore not only medical issues but also the legal, spiritual, and ethical aspects of dying in the ICU setting. Care providers are encouraged to approach death in a holistic manner and to find meaning in the deaths of their patients rather than to see death as a representation of failure. The authors are consistently mindful that (as Susan Block writes in Chapter 14) "Patients in the ICU challenge the clinician to both deny and attend to their humanity." Her statement reflects a theme of validation for clinicians who find themselves in a perpetual state of angst as they confront death in the ICU setting.

The editors state that their intention was to create a book that would be a practical resource, "dog-eared and scribbled on in nurses' stations and physician work rooms." If my copy of the book, littered with underscoring and Post-it notes, is any indication, that goal will be achieved!

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Topics in Palliative Care, Volume 4. Russell K Portenoy and Eduardo Bruera, Editors. New York: Oxford University Press. 2000. Hard cover, 288 pages, $59.95.

This fourth volume of **Topics in Palliative Care** has 2 things strongly in its favor, even before one starts reading. Portenoy and Bruera are well known in palliative care, so we assumed that they would involve informed writers and timely topics. Second, Oxford University Press has a strong track record of publications about palliative care. Different parts of this book will appeal to different audiences and will be applicable in different settings within palliative care. There are some real strengths.

The text is part of a series but certainly can stand alone as a reference. It is organized into 4 sections: survival estimation in palliative care, education and training in palliative care, procoagulation and anticoagulation therapy in palliative care, and issues in the assessment and management of common symptoms. Each of these sections has 3–4 chapters addressing different dimensions of these topics.

Most if not all patients who are confronted with a probably terminal illness want to know, "How long?" There are many reasons not to give a precise answer, the most important one being that it is only rarely that prognostication can be done with great accuracy, especially when death is not imminent. Regardless, that reason is used too frequently. Very often there is some idea about how long a patient will live. The patient and family are done an injustice if we do not share what we know. Section I, "Survival Estimation in Palliative Care," is meant to provide tools to help with this prognostication.

In Chapter 1, "Model for Estimation of Survival in Patients with Far-Advanced Cancer," the sample, and hence generalizability, is limited. The authors describe the research that led to development of their Palliative Prognostic Score. The authors rightly point out that clinicians remain reluctant to use yet another tool to supplement their clinical judgment in prognostication. The Palliative Prognostic Score does have utility as a research tool, but the use of a regression-based model in clinical practice is awkward at best. Numeric scores and quantitative analysis serve best when they have a didactic function. It is always up to the physician not only to integrate clinical data but also to weigh the importance of the data in the formation of participant decision making for a particular patient.

Chapter 2, "The Value of Symptoms in Prognosis of Cancer Patients," includes an extensive literature review of the relationship of certain symptoms, prognostic tools, or cancer type to life expectancy. The re-

view is thorough but there is little synthesis. The value of this chapter is greatest for the reader who has a specific question about, for example, what research has been done on quality of life and how it relates to survival. Clustering symptoms sometimes seems an attempt to reinvent the wheel. Symptoms vary so greatly that they often cannot be used to project prognosis, especially on the broader scale needed than the small subgroups described.

In Chapter 3, "Survival Estimation in Noncancer Patients with Advanced Disease," the authors did a useful job of placing palliative care in the context of reimbursement structures and definitional challenges. The authors' use of the SUPPORT data,[1] the acute physiology and chronic health evaluation (APACHE III), New York Heart Association data, and other prognostic tools that are commonly used give this chapter greater utility to clinicians. Their syntheses about heart disease, lung disease, and dementia are accurate and applicable across settings. Furthermore, the dimensions addressed are appropriate for discussion with the patient's family.

In Chapter 4, "Communicating a Poor Prognosis," the authors provide concrete tools that frame broad-based discussions with patients and families. The authors deal directly with the clinician's need (and, one might add, responsibility) to consider the patient's emotional responses. The questions and guidelines are realistic and useful for both beginning clinicians and those with greater experience. The authors integrate cultural considerations, the need not to overgeneralize those considerations, and the need to discuss in advance who will know what. These are all too rarely done, and the guidelines herein are realistic reminders.

Section II, "Education and Training in Palliative Care," contains practical information at a time when many palliative care services are starting, growing, or trying to prove their value in every sense of the word. Chapter 5, "Palliative Care Audit: Tools, Objectives, and Models for Training," is realistic in its recognition of the resistance to auditing by many caregivers. The authors provide useful tools and attainable goals and direction in the process and content of palliative care services. Their comments on a dozen tools provide a concise overview, and the assessments of utility are precise. The authors describe their use of that information in developing a new tool, the Palliative Care Outcome Scale (which is included) and its utility, seemingly broad, is discussed.

Education is sometimes viewed as the panacea to fix palliative care, or as the tool that would allow its optimal use. In Chapter 6, "Cancer Pain as a Model for the Training of Physicians in Palliative Care," the author provides a realistic description of why development and implementation are not just a matter of education. Using pain as a paradigm, the role (and sometimes intransigence) of attitudes and behaviors in the success and failure of educational efforts are portrayed well. Given that there is still no one widely agreed-upon definition of palliative care, the components described in this chapter (symptom control, communication/ethics, use of technology, patient/family, alternative care settings) are an interesting place to start the discussion of what constitutes palliative care. It's a logical extension into the much needed discussion of timing of palliative care interventions.

Chapter 7 is "The Palliative Care Unit as a Focus for Professional Education." The authors do an admirable job not only of drawing on their own considerable experience and expertise but also of establishing goals for hospice and palliative care programs at all levels of development, using international and historical considerations. Education objectives are clearly stated and realistic, addressing medical students, house staff, attending physicians, and visiting scholars (across disciplines).

Shifting the focus from setting to participants, Chapter 8 is "The Palliative Care Consultation Team as a Model for Palliative Care Education." Framed by marvelous literary references, the author of this chapter dives into the utility of a consultative model of palliative care. She also addresses most directly the need for the broadest definition of palliative care (quoting Portenoy) as pertinent across the disease trajectory. The model described is interdisciplinary and emphasizes the indispensability of including patient and family as team members. A symptom-based data collection process is described as a mechanism to document the value of the team.

Section III shifts to the issue of "Procoagulant and Anticoagulant Therapy in Palliative Care." Bleeding and emboli are frequent concerns in palliative care, and the question is often not just *how* to treat but *whether* and *how much*. A general goal should be to prevent thrombotic death. All too often, however, these therapies are used reflexively rather than placed in the context of palliative and end-of-life care. As with Section I, much of the information in Section III is for the subspecialist. Though the palliative care provider manages certain of these issues, the information in Section III allows the palliative care specialist to have a more informed conversation with the specialist who will most directly manage these therapies. The generalizability of the recommendations is limited, however, because all the chapters in this section focus on cancer patients.

In Chapter 9, "The Management of Bleeding in Advanced Cancer Patients," the authors describe risk factors and considerations in choosing whether and how to manage bleeding. And they go beyond physiologic considerations: local and systemic interventions are also described. Again, though some of these could be managed completely by a palliative care provider, most require further consultation with an oncologist or other specialist. This is especially true of those sections that provide guidelines that read "the optimal dose appears to be. . . " or "generally, such-and-such dose seems appropriate." The authors describe situations in which no further procoagulant or anticoagulant therapies should be used.

Management of thrombotic disorders is discussed in Chapter 10, "The Use of Heparin, Low Molecular Weight Heparin, and Oral Anticoagulants in the Management of Thromboembolic Disease." Again the focus is primarily on cancer patients. The authors present clinical trial results and tables to summarize data. They also describe factors to consider when choosing therapies. The advantages of low-molecular-weight heparin for short-term and long-term management are emphasized. The evolving nature of this field, however, will lead the clinician to seek a specialist for the most current recommendations and nuanced considerations.

Prevention is the focus of Chapter 11, "Prophylactic Anticoagulant Therapy in the Terminally Ill Cancer Patient." The author describes risk factors and provides general guidelines when considering anticoagulant therapy. Clinical trial data are provided. Again, appropriately, the authors include specific guidelines about when *not* to implement anticoagulant therapies.

Section IV, "Issues in the Assessment and Management of Common Symptoms," addresses not only problems with which providers grapple but also those of greatest con-

cern to patients and families. Chapter 12, "Intraspinal Analgesic Therapy in Palliative Care: Evolving Perspective," includes a good historical overview, and concise descriptions of delivery techniques. The intraspinal route is increasingly used and usually appropriately so. The challenge remains to recognize chronic pain as requiring a different paradigm of management than acute pain. The authors provide clinical considerations for choosing the optimal technique. Ideally this chapter will give the clinician the confidence to consult and include the expertise that the anesthesia and other teams can provide. Techniques such as nerve blocks and neurosurgical procedures are also mentioned.

Chapter 13 is "Pathophysiology and Assessment of Dyspnea in the Patient with Cancer." A sizeable portion of the chapter is devoted to pathophysiology; this serves as a sometimes necessary reminder about dimensions of dyspnea to be addressed with patients and families (even patients without cancer). The multidimensional nature of dyspnea is emphasized, though this is sometimes overlooked in clinical situations. The section on assessment is brief but helpful.

Chapter 14, "The Role of Oxygen in Cancer-Related Dyspnea," is a logical progression from Chapter 13. The author reviews the effects of oxygen on dyspnea, endurance, exercise tolerance, and other factors. No single recommendation is given about the use of oxygen to relieve dyspnea, but several physiologic mechanisms of how oxygen helps dyspnea are described.

The final chapter is "Treatment of Delirium at the End of Life: Medical and Ethical Issues." The discussion of medical management of delirium is thorough, and the synthesis and discussion on clinical implications are excellent. Still, too often in palliative care, clinicians focus only on symptom management as the basis for interventions. As a result, symptoms are sometimes treated without knowing the cause, and it is forgotten that by determining and addressing the cause (eg, drug interaction, nontherapeutic drug levels, certain electrolyte imbalances, medication adverse effects), multiple symptoms might be eradicated. The author of this chapter reminds readers that the causes of delirium (which is a highly prevalent condition at the end of life) must be determined when possible. The author also addresses language issues, exploring how "terminal restlessness" and other terms relate to delirium. The section on ethics includes a discussion on double effect. The ethical questions raised should be considered by clinicians confronting the end of a patient's life.

There is a range of clinical utility with this volume. More than half of the chapters were primarily or completely about issues pertaining to cancer patients. There is a strong research emphasis, which is a pivotal foundation for this still emerging field. Several of the chapters, however, lacked synthesis of the myriad findings presented or were concluded with a statement indicating that further research is needed. Though good for the student or researcher, this minimizes the applicability of some of the chapters. With prognosis, for example, what patients and families desire most is information about disease trajectory, rather than a prognostic date. Noncancer illnesses (eg, end-stage liver disease, congestive heart failure, multiple sclerosis) are by their natures episodic in the course of deterioration. The subsequent upswing makes prognostication more difficult but perhaps more important. Sometimes we really don't know, but we can often tell more than we do.

Palliative care is, or should be, interdisciplinary, and that team should include, as Abrahm writes in Chapter 8, the patient and family. Several of the authors reflect that idea; notably Higginson and Hearn (Chapter 5), Muir and von Gunten (Chapter 7), and Fainsinger (Chapter 15). Though some topics did not lend themselves to interdisciplinary considerations, others did, but the authors kept the focus on the physician as care provider and the patient as recipient of interventions. This somewhat restrictive view of the palliative care and end-of-life care teams would result in fewer resources for the patient and family.

The editors succeeded in including leaders in the fields, and the book offers diverse perspectives and beliefs about palliative care. There are some important strengths, precisely for the reasons we expected: the editors' understanding of where the field has been, its current struggles, and where we will go. This book provides insight to how we will get there.

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REFERENCE

1. Knaus WA, Harrell FE Jr, Lynn J, Goldman L, Phillips RS, Connors AF Jr, et al. The SUPPORT prognostic model. Objective estimates of survival for seriously ill hospitalized adults. Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments. Ann Intern Med 1995;122(3):191–203.

Textbook of Palliative Nursing. Betty Rolling Ferrell PhD and Nessa Coyle RN MS NP, Editors. New York: Oxford University Press, 2001. Hard cover, illustrated, 854 pages, $75.

This is an excellent reference book for nurses providing palliative care in the hospice and acute care settings. There are 80 contributing authors from around the world, a fact that I thought could have made the book fragmented. However, the editing is so well done that the chapter-to-chapter segues are seamless. The book is divided into 9 parts and 64 chapters. Each chapter has an extensive bibliography from a wide variety of journals and other sources. I was able to find a couple of typographical errors but they were not distracting.

Each chapter begins with a quote from a patient, family member, or health care professional that provides an illustration of the importance or relevance of the topic. Some of the quotes are quite moving, as they illustrate the dying person's perception of the dying experience and capture the essence of what was important to him or her.

The chapters are all organized in a similar manner. Each opens with a definition of the problem that is outlined in the chapter title. In "Bowel Management: Constipation, Diarrhea, Obstruction, and Ascites," for example, one knows exactly what one is about to read! After the definition of the problem, the physiology and pathophysiology are described. The chapter then proceeds with the assessment and management of problems, using very descriptive and detailed case studies to illustrate creative solutions. What I liked best about the case studies was that they were not used to neatly illustrate points. Most difficult patient problems do not have neat solutions in real life, and frequently case examples are too one-dimensional to really be helpful in a practical sense. These case examples go through the first-line solution to the problem, providing a second-line and even a third-line solution to the problem when previous at-

tempts are not successful. Some case examples illustrate that not all problems can be solved. The one I remember best was about an alcoholic patient with a terminal illness who did quite well with gaining weight while in the hospital but became cachectic upon returning home. The patient died at home sooner than expected, still drinking. That example seems based in reality and hence is more useful to the reader.

For most physical symptoms a pharmacologic approach is given, as well as a nonpharmacologic approach. This is really helpful, because some patients may respond better to the nonpharmacologic method, or a combination of both types of therapies may yield the sought-after result. I found all chapters to be very inclusive of potential solutions to complications related either to the dying process or to the treatment of the underlying disease. The process of trial and error was illustrated time and again, which made very clear the idea that palliative nursing is about problem-solving for the patient and family. Throughout the book, emphasis is on the patient and family, because so many times the caregiver most affected by the symptoms experienced by the patient is a family member.

The book is written in an easy-to-understand way. It is very practical and also very detailed. For instance, in the chapter entitled "Dysphagia, Dry Mouth, and Hiccups" there is a very thorough description of artificial saliva. I learned that mucin-based saliva substitutes are better tolerated than those derived from carboxymethylcellulose. And both are better tolerated as a spray than as a gel or rinse. That level of detail is present throughout the book for every symptom a dying person is likely to experience. A real treat for me was reading a recipe for the milk and molasses enema I have heard about but never seen used. I was surprised to learn that the recipe calls for powdered milk.

Solutions for physical symptoms are just one facet of this book. In the section on psychosocial aspects of dying there are excellent sections about communication traps, the meaning of hope, how to support family members caring for the dying person, and planning for the time of death. I especially liked the analogy used in describing how to communicate when it is necessary to switch from curative therapy to palliation; they call it "making a lane change" and further discuss it in terms of anticipating roadblocks, timing transitions, and shifting gently. It is an analogy that most can relate to and very effectively shows what kind of techniques should be avoided when the patient reaches the stage in the disease when a change is necessary.

The section on pain assessment is superb. The creative approach to providing pain relief while allowing the patient to be alert demonstrated how palliative nursing really addresses the needs of the patient and the family. Many families are distressed by the somnolence experienced by the dying patient when the patient takes enough narcotic to ease the pain. The multifaceted approach to pain management involves the use of narcotics with both long-acting and short-acting characteristics, as well as using nonsteroidals for somatic pain. The chapter conveys the story of a patient whose somnolence made it so that he could no longer go fishing with his son, which had been a source of great pleasure for him, and how, after working with the palliative care nurse on the problem of pain management, the patient had superior pain control, was no longer sleepy all the time, and so was able to engage in activities that brought him pleasure. There are several pain assessment tools described in this chapter, and the one on assessing pain in the nonverbal patient was particularly helpful.

The chapter on nausea is also outstanding, with an excellent table (covering 2 pages) that lists a wide variety of anti-emetics, their indicated use, dose, route, schedule, adverse effects, and a section for comments, which has information about using the drug with caution in hepatically or renally compromised patients as well as specific information about drowsiness and which population might find the drug most useful. The section on nonpharmacologic interventions was quite extensive. Techniques described include biofeedback, self-hypnosis, relaxation, distraction, acupressure, and music therapy. Involving the family in providing those therapies is emphasized. The take-home point in this section, as in other sections, is on helping the patient maintain as high a quality of life as possible for the time he or she has remaining.

Besides being an excellent reference for patient-focused symptom management, the book is an outstanding resource for anyone interested in the pitfalls of palliative nursing. The politics of referral and third-party payment have a very negative effect on patient outcome. Referred to hospice too late, many patients never reap the benefits of a multidimensional health care team. The reasons for late referral are discussed. The effect of palliative nursing on the individual nurse is also discussed, and the timeline for professional development is described. The fact that burnout occurs if the nurse cannot surmount certain professional challenges is described, but I was not satisfied with the discussion of preventing burnout or intervening once it has occurred. In some chapters the authors describe the role of the palliative care nurse with evangelistic fervor, stressing the nurse's role over other professionals. At times, that seems to diminish the role of other members of the health care team, which, since it is a book aimed at nurses, seems to be preaching to the choir.

If pressed to find a weakness in this book, I would point to the inconsistent quality of the illustrations. Some graphs are too "busy" and difficult to read. Some are just too simplistic to be helpful. Others were the backbones of their chapters. The poor quality illustrations could have been discarded.

Overall this is an excellent, well-written book that is packed with extremely helpful information. It is full of practical solutions to common patient situations and yet devotes a substantial amount of time to political, historical, and international perspectives on palliative nursing. I believe it would be very instructional to a nurse new to palliative care and would also serve as an excellent resource for the experienced palliative care nurse.

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Final Victory: Taking Charge of the Last Stages of Life, Facing Death on Your Own Terms. Thomas A Preston MD. Roseville, California: Forum/Prima Publishing. 2000. Hard cover, 252 pages, $22.95.

Death and dying and end-of-life issues have generated much discussion and debate among ethicists and health care professionals for more than 2 decades; however, in recent years, these topics have moved to the front burner in our nation's public consciousness. This is a consequence of an explosion of "rights" issues over the past 40 years: the civil rights, human rights, anti-war, and en-

vironmental movements, and the Americans with Disabilities Act, to name just a few. And now the baby boomers—the generation that championed those movements—are aging and are faced with their parents' illnesses and deaths, as well as their own mortality. They are thinking, talking, and writing about their experiences and about how it should be and who should decide. It was only a matter of time before the rights of the dying hit the radar screen.

Yet anyone who has counseled a patient or family member on end-of-life issues knows that most people are uninformed, lost, and afraid. They are unprepared for death and the decisions surrounding end-of-life care, yet have a strong desire and need to remain in control. To do that they must understand the treatment options, identify their goals, and know how to achieve them.

Dr Preston has written an important book that will help patients and families who are trying to navigate the maze of end-of-life care.

Final Victory is a thoughtful and comprehensive resource that will enable patients and their families and friends—anyone and everyone—to prepare for and take control of the last days of life. Health care professionals in particular would be wise to read this book and incorporate its lessons into their practice.

Dr Preston organized the content along a trajectory from the thinking and planning stages of advance directives; through the diagnosis, prognosis, and treatment stages; to the options for end-of-life care and saying goodbye. He defines and explains the often-confusing terminology of end-of-life options—resuscitation, withdrawal of care, physician-assisted suicide, aggressive comfort care, and terminal sedation—and gives advice on how to discuss those and related matters with physicians and family members.

In Part One, "Taking Charge from the Start," Dr Preston provides a foundation for later exploration of specific issues and suggested strategies. He introduces the terminology of illness and dying and effectively grabs the reader's attention with an anecdote about a patient who had suffered a debilitating stroke that had left her unable to communicate. Family members watched her in that condition for 4 years, knowing that it was not what she would have wanted. They were sad and depressed and felt responsible for her being in that condition.

In language accessible to a lay audience, Dr Preston explains how modern medicine and new technologies have altered the way we die, so that a terminally ill patient may be given antibiotics for pneumonia or kidney dialysis for renal failure, thereby prolonging the dying process. He explains that some patients could benefit from the additional time to be with loved ones and to say goodbye, whereas for others it may be an exercise in futility that just prolongs the pain and suffering of dying.

Dr Preston stresses that it is important for patients to discuss their thoughts and beliefs about end-of-life care with their physicians and family members in order to lay the groundwork for future decision-making. He also advises patients to ascertain if the physician is open to considering comfort measures and what he terms "assisted dying" or "aid in dying" if and when treatment is no longer beneficial.

Part Two, "Taking Charge After the Diagnosis," focuses on becoming informed about the disease process and recognizing that the goal of most physicians is to cure and prolong life. Dr Preston suggests that the clinical momentum of aggressive care can be very difficult to stop. He provides practical and invaluable direction on how to speak with physicians about the disease, prognosis, and treatment plan. Dr Preston states that when asking the doctor about the prognosis and what to expect along the way, the doctor may be vague and explain that the course of the disease is variable and unpredictable. He suggests that reframing the question may be more effective; for example, "From your experience, doctor, describe the average course of this disease from now until the end." (Page 86)

Knowing what to expect and how to ensure a peaceful death are covered in Part Three. Two chapters in particular are especially comprehensive and thoughtful. In "Strategies for Peaceful Dying," Dr Preston describes 3 principal medical methods for achieving a peaceful death: keep the dying phase short, get as much comfort care as possible to eliminate symptoms, and select a location for the last days. The chapter "Arriving at the End" offers family and friends advice on what they can do to help and how to respond to distressing symptoms such as intractable pain, loss of appetite, and hallucinations. How to say goodbye and the importance of asking for and offering forgiveness are also addressed.

Specific information can be easily accessed. Each of the 3 parts begins with a list of topics to be covered, and every chapter ends with a summary list of important facts and considerations. The chapter titles are specific and make it easy to find particular information. The index is detailed and cross-referenced, and there are 2 appendixes: one containing sample advance directive forms, the other listing organizations and Internet sites where more information can be found. There is a nice section on "suggested reading," but there are no references within the text, despite the fact that Dr Preston occasionally cites studies and statistics.

Despite that good accessibility, the book could have been better organized. Several topics are discussed in numerous chapters, including communicating with important people, discussions of end-of-life options, and aid in dying. Although covered in the context of different stages of illness, this was repetitive and caused some confusion. Resuscitation is described in Chapter 2 as an electrical shock. In Chapter 8 it is explained that resuscitation is often called cardiopulmonary resuscitation or CPR and that it refers to procedures done to restart the heart. End-of-life treatment options are discussed at least twice: in Chapter 2, "Medically Managed Dying," and again in Chapter 8, "The Options of Assisted Dying."

The book was most problematic when it came to the subject of physician-assisted suicide. Dr Preston gives this considerable attention, despite stating that aggressive comfort care and terminal sedation are almost always adequate to control pain, and physician-assisted suicide is legal in only one state, Oregon. Certainly physician-assisted suicide is a topic that must be covered, but Dr Preston appears to be advocating its wider use. In a special section devoted to physician-assisted suicide he writes that, although it is illegal, it does occur and that "the majority of physicians and adults in this country believe dying patients should have some access to assisted dying." This is rhetorically problematic because it suggests that most people support physician-assisted suicide, when in fact, "assisted dying" is a much broader category of options. Putting those phrases together in one sentence is highly misleading. Furthermore, such an ideological tangent in a reference or guidebook undermines the credibility of the book as a whole for those who disagree with the author's point of view. Physician-assisted

suicide is a sensitive and contentious issue, and advocating for either side could be alienating to some readers.

In sum, **Final Victory** is a welcome and much needed resource. Health care professionals who avail themselves of this quick read will be better informed, more understanding of patient and family needs, and therefore better prepared and more adept at communicating with patients and families about end-of-life care. Patients and their loved ones will find comfort in their ability to frame the questions, consider the options, and maintain control, as they make their way through the end-of-life labyrinth to a peaceful end.

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Chronic Lung Disease in Early Infancy. Richard D Bland and Jacqueline J Coalson. (Lung Biology in Health and Disease, Volume 137. Claude Lenfant, Executive Editor.) New York: Marcel Dekker. 2000. Hard cover, 1,062 pages, $235.

Chronic Lung Disease in Early Infancy is an exhaustive consolidation of clinically relevant information and basic science and biomedical research data available on the topic of infant chronic lung disease (CLD), an illness previously called bronchopulmonary dysplasia. This text expands from the historical perspective and epidemiology of infant CLD to diagnostic criteria and therapeutic modalities to decrease both the risk and severity of the illness. Care of the infant from prenatal through early childhood is discussed. Figures showing chest radiographs and pathology tissue sections of lung parenchyma provide visual images of the disease process. The clinical section is followed by an organized, thorough discussion of the advances in basic and applied biomedical research in this field. This text is therefore of value not only to those providing health care to neonates at risk for or who actually have CLD, but also to those beginning or actively pursuing research on this topic. The targeted audience includes not only neonatologists, pulmonologists, critical care nurses, and respiratory therapists, but also laboratory research personnel. The depth of the book is too great for those early in their clinical training, but it would serve as an excellent reference for house staff officers. The combination of clinical and compiled research data would be optimal for someone subspecializing in this field, since the text is a comprehensive collection of well referenced knowledge on infant CLD. In addition it clearly defines questions that are as yet unanswered.

This sturdy, hard cover book is well organized into 5 parts: Clinical Aspects; Normal and Abnormal Alveolar and Airway Development; Normal and Abnormal Development of the Lung Circulation and Interstitium; Mechanisms of Lung Injury and Repair During Development; and Models of Lung Injury and Repair During Development. Most of the clinically relevant material in the text is in the first part. The other 4 parts focus on research developments. Each part is comprehensive and independent of the other parts, allowing one to focus on his or her particular subject of interest, but having more information easily available should they want to expand. The table of contents lists not only the chapters composing each part but also the contributing sections of each chapter in an outline format. Identifying areas of interest was therefore quite easy. Both an author and a subject index are included.

The editors did an excellent job of organizing the work of the 75 contributing authors. Each part of the book is independent of the other parts and may be read without reference to the others, which causes some repetition between the parts and even among chapters within the same part, but only rare discrepancies. The text was extremely well referenced, and I was impressed with how comprehensive it was.

Because of my medical background and clinical interest I focused on Part I. However, the clear writing in the other parts made the research material easy to comprehend and follow. The distinguished reputations of the contributing authors, the completeness of the content, and the thorough referencing, however, makes the text appealing to those with established research backgrounds.

Part One begins with the historical perspective and epidemiology of infant CLD. It expands to preventive and therapeutic strategies, including ventilatory management and pharmacologic and nutritional support. The chapters on effects of infection on the disease process, role of inflammatory mediators, radiographic features, and pathology of CLD of early infancy are examples of how the text includes knowledge from multiple subspecialties involved in the care of these infants. Part One covers not only prenatal and neonatal intensive care unit treatment issues but also follow-up care during infancy. Data from studies of young adults who once had bronchopulmonary dysplasia complete Part One.

With new therapies having substantial impact on the prevention and severity of the illness, CLD of early infancy is obviously an evolving disease process. The authors did a superb job of making a clear distinction between "old" CLD (which was much more common before the advent of our current interventions with pharmacologic therapies and improved ventilation strategies) and "new" CLD, aspects and prognosis of which are more important to current clinical practice.

Editors Bland and Coalson are very specific in outlining their goals in the preface:

> 1. Present the important clinical and pathological features of a disease that represents a major cause of long-term hospitalization, slow growth, and recurrent respiratory ailments in early childhood. 2. Provide a timely, comprehensive review of what is known about lung development, injury, and repair as they might relate to the pathogenesis of CLD of early infancy. 3. Define what relevant information needs to be learned and how we might learn it. 4. Relate this information to potential therapeutic and preventive strategies.

Those goals were clearly accomplished. This text will serve as an excellent reference to those active in research or providing health care to infants. Health care providers should be aware that the text's clinically relevant information is mostly limited to the first part of the text. The text may be too exhaustive and abstruse for those early in their clinical training.

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Sleep Apnea: Implications in Cardiovascular and Cerebrovascular Disease. T Douglas Bradley and John S Floras, Editors. (Lung Biology in Health and Disease, Volume 146, Claude Lenfant, Executive Editor.) New York: Marcel Dekker, 2000. Hard cover, illustrated, 554 pages, $195.

The past decade has witnessed substantial growth in research and better understanding of mechanisms in the field of sleep medicine. One important subject that has received particular attention is the association between cardiovascular diseases and sleep apnea. This stems from the high prevalence of both cardiovascular disease and sleep apnea in the general population. Because of the multidisciplinary nature of sleep medicine, research has been performed by seemingly disparate groups of investigators in different areas of medical research. Although there are several well-written general textbooks on sleep medicine, Bradley and Floras have assembled 32 authors in basic and clinical investigation to create this book, which is the first of its kind addressing the current state of the art in sleep apnea. The authors are internationally known leaders in their fields, and the editors should be commended for organizing such a diverse group of writers. The book is the 146th volume of the series Lung Biology in Health and Disease, supported in part by the National Institutes of Health. The chapters are grouped into 4 parts: Influence of Sleep and Respiration on the Cardiovascular System; Sleep Apnea and Hypertension; Sleep Apnea, Ischemic Heart Disease, and Cerebrovascular Disease; Sleep Apnea and Congestive Heart Failure.

The first group of chapters reviews basic mechanisms of regulation of cardiovascular function, baroreception/chemoreception, and the interactions of sleep and the respiratory and cardiovascular systems. The first 2 chapters deal with the influence of respiration on autonomic regulation of heart rate and blood pressure. These chapters cover the complex and intricate processes that govern the interaction of respiration with cardiovascular regulation and the role of chemoreflex-baroreflex interactions. Chapter 3 deals with the neural mechanisms of the cardiac and respiratory system and the role of the sympathetic nervous system. Chapter 4 discusses the mechanical interactions between the respiratory and circulatory systems and the influence of intrathoracic pressure on cardiac performance. Chapters 5 and 15 deal with the effects of sleep on the cardiovascular system and the stimulatory effects of arousal from sleep on heart rate and blood pressure and transient uncoupling of the baroreflex. Chapter 6 deals with the influence of sleep apnea on autonomic control of the cardiovascular system and effects of hypoxia and hypercapnia on the sympathetic response. Chapter 7 examines in detail the mechanism of acute and chronic blood pressure elevation in animal models of obstructive sleep apnea. Chapter 8 focuses on the role of chemoreceptors and sympathetic activity in chronic hypertension in the rat—a model that has provided substantial understanding about the cardiovascular response to intermittent hypoxia. Chapters 9–11 deal with acute hemodynamic responses to sleep apnea, primarily in humans.

Chapter 12 presents the epidemiologic links between sleep apnea and sustained hypertension and cardiovascular disease, and convincingly describes a significant association, if not causation, between sleep apnea and sustained hypertension. Similarly, Chapter 13 discusses the association between cerebrovascular disease and sleep apnea. There is extensive coverage of sleep apnea and congestive heart failure in Part Four, in which the prevalence, pathophysiology, and treatment of sleep apnea in relation to heart failure are extensively discussed. Chapter 20 would be of potential interest to respiratory therapists in terms of treatment options and particularly the role of continuous positive airway pressure in patients with congestive heart failure and sleep apnea. This exciting field of research is rapidly advancing and would be of immediate clinical application.

The subject index is well organized. All the first authors mentioned in the text are listed in the author index. This provides a useful guide to the contributions of investigators on this subject.

The chapters are well written and are detailed enough to provide background for further investigations. However, some of the chapters are difficult to follow and thus will often require supplementation with original articles. This problem is essentially due to the complexity of the topics rather than the style of the authors, and is overcome with an extensive and up-to-date bibliography in each chapter. The book is written primarily for scientists and clinical investigators of cardiovascular and respiratory regulation during sleep and the effects of sleep apnea on those systems. The book is an excellent resource with its comprehensive bibliography. However, it is not intended as a clinical manual.

I commend the editors on their efforts in compiling current understanding on this important topic and potential avenues for future research.

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The White Death: A History of Tuberculosis. Thomas Dormandy. New York: University Press, 2000. Hard cover, illustrated, 433 pages, $29.95.

Readers may wonder about the difference between "sanitarium" and "sanatorium," without ever feeling a need to open a dictionary to find out. The differences in meaning and usage of these words may be interesting and useful, but more as footnotes and background than as important elements in the contemporary practices of medicine and public health. As Thomas Dormandy notes in **The White Death: A History of Tuberculosis**, "sanatorium," based on the Latin verb *sanare* (to cure) was favored by the German founders of the sanatorium movement, implying active medical intervention and is the term more often used in Europe, whereas "sanitarium," based on the Latin noun *sanitas* (health) was preferred by American practitioners, in the belief that the benefits came from healthy living. As a fairly typical illustration of the American usage, a small mountain near Boulder, Colorado, was named "Sanitas" after the tuberculosis (TB) sanitarium at its foot, which has since been converted to a modern hospital. Dormandy's **The White Death** is similarly interesting and useful, but more for the person who is inclined to open a large dictionary to learn the derivation, earliest usages, and precise meaning of a word than for one whose dictionary use is limited to the spell-check function of a word processor.

The White Death offers a leisurely and conversational review of the historical impact of and fight against TB in Europe and the United States. The book falls short of the global implication of its subtitle, but satisfies a more limited—though unstated—goal very nicely, with interdisciplinary breadth, humor, and pathos. Tremendous pa-

thos, unfortunately, is one of the main themes of this disease, as shown by the details of loss and sadness in the title and on almost every page of the book. The disconnection between the title and the content of the book, moreover, highlights and magnifies the cruelty of this disease by not fully addressing its global severity and impact. A different subtitle, such as "A History of TB in Western Civilization," would be more precise and would acknowledge the fact that this book pays little attention to most of the world. Unfortunately, in most of the world, where TB kills 2 million individuals each year, it remains far more than the footnote that it seems, deceptively, to have become for many in Europe and North America.

The White Death extensively chronicles the impact of TB on Western culture, and with a broad-minded perspective. Dormandy presents stories of the disease as it affected prominent artistic and political figures and families, and adds a liberal dose of personal commentary on the social and intellectual context as well as occasional forays into criticism of art and literature. For example, Aubrey Beardsley's 1896 drawing *Death of Pierrot* is the frontispiece. It shows the emaciated but still delicate clown lying in bed wearing frills, while costumed visitors tiptoe into the room holding fingers over their lips. Dormandy describes this drawing as Beardsley's drawing of himself on his death bed. He goes on to suggest that Beardsley may have been the model for the artist Louis Dubedat (who is dying of TB) in George Bernard Shaw's play *The Doctor's Dilemma*. He goes on to sketch Shaw's "gallery of medical luminaries, caricatures but so real that they are instantly recognizable even today," who gather around the artist. He also quotes briefly from the play, citing Dubedat's death scene as, "moving for... the sheer defiance of his last great speech and Bohemian creed—and for its truth." The quoted passage begins and ends: "I'm perfectly happy. I'm not in pain.... I believe in Michael Angelo, Velasquez and Rembrandt; in the might of design; in the mystery of colour; in the redemption of all things by Beauty everlasting, and the message of Art that has made these hands blessed. Amen."

The chapter on "The Romantic Image" ends dramatically with another Beardsley drawing, "drawn between bouts of pulmonary haemorrhage," of the ancient Greek feminist and peace activist Lysistrata, "showing the lady decorating an adoring giant phallus." "Impotent in the terminal stages of his illness, his drawings became increasingly obscene while reaching their peak in elegance and economy." These latter comments on Beardsley come in Dormandy's chapter on DH Lawrence, "The Undying Man," in the context of several pages on the influence of TB, similar to their influence on Beardsley's work, on the writing of *Lady Chatterley's Lover*. He describes a creative life of the mind that overcompensates for progressive physical wasting and impotence.

Dormandy goes so far in his criticism as to extend the use of the word "tuberculous" from the patient to the work of art. Robert Schumann is described in a way that may hold a special resonance for respiratory therapists: "For most of his marriage [to Clara, who died from a massive pulmonary bleed 18 months before he died] he was in fact ailing, filling a whole cup with horrible purulent phlegm every morning... " A late poem that Schumann wrote to his children is described as "one of the most memorable tuberculous farewells." It includes the lines, as translated by Dormandy, "And tell me, where is your mother? 'Mother is resting.' —That is what I want to do too: I am tired, my dears./Therefore farewell!"

After several chapters on the sanatorium movement, **The White Death** includes a particularly effective chapter on Anton Chekhov, "Doctor, Patient, Writer." This chapter combines personal and medical biography with literary criticism. For Dormandy, Chekhov's plays are

> unique insider studies of the tuberculous destiny without ever once mentioning the illness... which struck down the young and crippled and shortened their physical but often enriched their spiritual life, which excluded them from many of the more intensely pleasurable pursuits yet made them abnormally responsive to some of the mundane beauties and sadnesses of the world around them. Most surprisingly perhaps, it undermined the bedrock of most young people's daily existence, the planning for a future, and could yet sustain unquenchable hope.

This passage is typical of Dormandy's thoughtful and conversational style, in his frequent combining of specific instances with broad generalizations.

Dormandy describes Chekhov's play *The Three Sisters* as "his most profoundly tuberculous." He writes that even though neither the audience nor the actors might be "aware of the pervasive presence of a physical illness" in a production of the play, the lives portrayed "only make sense on the assumption of some insuperable underlying though unstated physical impediment." He goes on briefly to analyze the play from that perspective, as well as to describe how the other plays also "recreate the tuberculous existence of Chekhov's days more truly than any other work of art or literature." This approach to medical literary criticism may give readers a useful addition to the perspectives they bring to a written text or drama.

Although Dormandy waxes most lyrical and idiosyncratic in his chapters on cultural figures affected by the disease, he devotes approximately equal space to the development of the science of TB, telling stories that may be more familiar to medical readers, but with a depth and with citations that may serve as useful reference material. These stories range through some of the major landmarks of Western medical history, showing how important this disease was in the development of modern medicine. In addition to describing the contributions of the well-known giants of medical history, he also very nicely describes some of the lesser-known contributors. For example, in the chapter "Three Heretics" he describes "Dr George Bodington (1811–1882) one of a species endangered even in his day and even more today, an independent-minded general practitioner removed both geographically and in spirit from the fashionable centres of learning, impatient with pontifical pronouncements handed down from on high and skeptical about medical dogmas based on revelation." His 1840 "Essay on the Treatment and Cure of Pulmonary Consumption: On Principles Natural, Rational and Successful" castigated the then-standard practices of treating TB with digitalis and tatar emetic and enclosing patients in sealed rooms. He advocated an early version of the sanatorium (Dormandy's preferred form of the term), for which he rented a house close to his home, "for patients 'who are desirous or who are recommended to remove from their homes for the benefit of the dryness, freshness and purity of the air, constant and watchful care, exercise and a varied and nutritious diet.'" The only drugs he used were small quantities of wine with meals and morphine as a sedative. Although he was in turn castigated and then ostracized by the medical establishment, perhaps because of the

confrontational vehemence of his presentations, in addition to having an idea that was ahead of its time, the approach he described subsequently became the standard of care for treating TB for a nearly a century. In a footnote Dormandy includes Shaw's reference to Bodington in *The Doctor's Dilemma*, which was written well into the sanatorium era: "He was ruined and driven out of practice for only opening the windows: and now we won't let a consumptive patient have as much as a roof over his head." Here, as elsewhere in the book, Dormandy's footnotes are as interesting and pertinent as is the main text.

Dormandy gives colorful descriptions of the discovery of the tubercle bacillus, the origins of radiology, the development of the bacillus of Calmette and Guérin (BCG) vaccine, the evolution of thoracic surgery, and the other landmarks in the science of TB. These well-referenced stories include social and biographical commentary and criticism as well as connections to the cultural figures affected by those medical advances. He describes how George Orwell, for example, was able to take advantage of well-placed and wealthy connections to obtain a supply of streptomycin in 1947, before this revolutionary new TB drug became widely available, and how it helped to extend his life long enough for him to complete his work on the book *1984*. Unfortunately, he developed a hypersensitivity reaction to the drug (well-described by Orwell in a letter that Dormandy quotes at some length) and did not live long enough to learn about the other new antibiotics, para-aminosalicylic acid and isoniazid, that were discovered to be effective against the disease at about the same time.

The White Death includes a final chapter, "An Imperfect Civilisation," that introduces and confronts some of the contemporary and global issues in TB, such as its interaction with the human immunodeficiency virus pandemic, multi-drug resistance, and directly observed therapy. Dormandy points out and briefly outlines some of the tremendous medical, political, and cultural complexity of these modern challenges. He concludes with a touching ending that refers back to earlier parts of the narrative, in which he reminds modern readers of the folly of being overly dogmatic or impatient for a final practical approach or cure, but at the same time describes how persistent hope is part of the "personality" of TB.

Among American TB controllers one realization of the past decade has been that TB never was brought under control in most of the world, along with the recognition that only by controlling TB throughout the world will the disease really come under control here. **The White Death** invites a sequel that would extend its history into other parts of the world—into other places and cultures. Reading about how creative lives were cut short by TB during the Romantic period of Western culture, for example, one wonders about the creative—as well as the ordinary—personalities of other places and cultures that have been and that continue to be cut short in such large numbers. Perhaps an equally compassionate history of TB outside of western civilization is already now being written.

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AARC Clinical Practice Guideline

Pulmonary Rehabilitation

PR 1.0 PULMONARY REHABILITATION:
Pulmonary rehabilitation is a restorative and preventive process for patients with chronic respiratory disease.

PR 2.0 DESCRIPTION/DEFINITION:
Pulmonary rehabilitation (PR) has been defined as a "multi-disciplinary program of care for patients with chronic respiratory impairment that is individually tailored and designed to optimize physical and social performance and autonomy."[1]

As lung reserve declines, dyspnea worsens and independent daily activity performance erodes. PR provides multidisciplinary training to improve the patient's ability to manage and cope with progressive dyspnea.[2]

Although PR efforts are often focused on patients with chronic obstructive pulmonary disease (chronic bronchitis and/or emphysema),[3-6] other conditions appropriate for this process include, but are not limited to, patients with asthma,[7] interstitial disease,[8] bronchiectasis,[8] cystic fibrosis,[9-11] chest wall diseases,[8] neuromuscular disorders,[12,13] ventilator dependency,[14,15] and before and after lung surgery for transplantation,[16] volume reduction,[17,18] or cancer.[19,20]

PR services include critical components of assessment, physical reconditioning, skills training, and psychological support.[2,21] Additional PR services may include vocational evaluation and counseling.[22] The PR program must be tailored to meet the needs of the individual patient, addressing age-specific and cultural variables, and should contain patient-determined goals, as well as goals established by the individual team discipline.[20,23] Both patients and families participate in this training administered by health care professionals. These pulmonary rehabilitation services are overseen by a medical director to assure appropriate performance by the program staff and to assure proper service delivery.[2]

This guideline is appropriate for pediatric, adult, and geriatric patients in whom clear indications for rehabilitation are present and who possess the necessary cognitive and physical capabilities.

Based on the individualized assessment the following areas of education and training should be considered:[2]

- **2.1** pulmonary anatomy and physiology including the pathophysiology of lung disease[24-26]
- **2.2** description and interpretation of medical tests[27-33]
- **2.3** bronchial hygiene techniques[34,35]
- **2.4** exercise conditioning and techniques that include:[36]
  - **2.4.1** breathing retraining[37]
  - **2.4.2** endurance, strength, and flexibility training
    - **2.4.2.1** upper extremity[37-42]
    - **2.4.2.2** lower extremity[37,41]
  - **2.4.3** ventilatory muscle training (its role is still undetermined, since no evidence exists that it contributes to functional improvement when added to a traditional upper and lower extremity exercise training program).[1,36]
  - **2.4.4** energy conservation as it applies to activities of daily living[43,44]
- **2.5** indications, actions, and side-effects of medications including non-prescription products, such as vitamins, over-the-counter medications, and herbal remedies[6]
- **2.6** functional self-management
  - **2.6.1** self assessment and symptom management[45]
  - **2.6.2** infection control with emphasis on avoidance, early intervention, and immu-

nization[46-48]

2.6.3 environment control

2.6.4 indications for seeking additional medical resources

2.7 sleep disturbances, eg, insomnia and sleep apnea as they relate to chronic lung disease

2.8 sexuality and intimacy[49,50]

2.9 nutrition[51-54]

2.10 smoking cessation[55-57]

2.11 psychosocial intervention and support[21,58]

2.12 available community services, including patient/family support groups[59]

2.13 advance care planning[60,61]

2.14 travel issues[62]

2.15 recreation/leisure activities[63]

2.16 stress management

2.17 indications for oxygen, and methods of delivery[64]

PR 3.0 SETTINGS:

PR may take place in, but is not limited to:

3.1 the inpatient setting, including medical center, skilled nursing facility, or rehabilitation hospital[2]

3.2 the outpatient setting[2,65]

3.2.1 outpatient hospital-based clinic

3.2.2 comprehensive outpatient rehabilitation facility (CORF)

3.2.3 physician's office

3.2.4 alternate or extended care facility

3.2.5 patient's home[65]

PR 4.0 INDICATIONS:

The indications for PR include the presence of respiratory impairment potentially responsive to the techniques available.[1,2,36] Such impairment may be manifested as:

4.1 dyspnea experienced during rest or exertion

4.2 hypoxemia, hypercapnia

4.3 reduced exercise tolerance or a decline in the patient's ability to perform activities of daily living

4.4 an unexpected deterioration or worsening symptoms against a background of long-standing dyspnea and a reduced but stable exercise tolerance level

4.5 the need for surgical intervention (pre- and postoperative lung resection, transplantation, or volume reduction)

4.6 chronic respiratory failure and the need to initiate mechanical ventilation

4.7 ventilator dependence

4.8 increasing need for acute care intervention, including emergency room visits, hospitalizations, and unscheduled physician office visits

PR 5.0 CONTRAINDICATIONS:

The initial assessment of the patient should establish his or her willingness to participate in the rehabilitation process. The presence of certain conditions would make successful completion of the rehabilitation process unlikely.[2]

5.1 Potential contraindications to PR include ischemic cardiac disease, acute cor pulmonale, severe pulmonary hypertension, significant hepatic dysfunction, metastatic cancer, renal failure, severe cognitive deficit, and psychiatric disease that interferes with memory and compliance. The decision to provide or withhold PR should be based on a thorough, individualized assessment.

5.2 Substance abuse without the desire to cease use would seriously interfere with successful PR.

5.3 Physical limitations such as poor eyesight, impaired hearing, a speech impediment, or orthopedic impairment may require modification of the PR setting but should not interfere with participation in a PR program.

PR 6.0 HAZARDS/COMPLICATIONS:

Hazards/complications associated with PR are primarily related to the exercise program. During exercise the cardiovascular and ventilatory systems must be able to respond to increased demands. Exercise can lead to muscle or ligament injuries.

PR 7.0 LIMITATIONS OF METHOD:

7.1 Patient related

7.1.1 The patient may have a disease process that has progressed to the stage where rehabilitation is not possible.

7.1.2 The patient may not adhere to or complete the program because it appears to be complicated or because of a sense of hopelessness, depression, or a lack of motivation.

7.1.3 The patient/patient family may be reluctant to make changes in their usual program, medications, start new therapy,

quit smoking, use supplemental oxygen, or exercise.[23]

7.1.4 There might be concerns or limitations in transportation.

7.1.5 Financial resources might not be available.

7.1.6 The patient may have to stop the program because of an acute exacerbation, or worsening of another medical condition.

7.2 Related to the health care system

7.2.1 Reimbursement by intermediaries or third-party payers is not standardized.

PR 8.0 ASSESSMENT OF NEED:

8.1 The patient must be under the care of a physician for the pulmonary condition for which he or she needs rehabilitation. Appropriate members of the PR team participate in the patient's assessment. The initial evaluation should include the medical history, diagnostic tests, current symptoms, physical assessment, psychological, social, or vocational needs, nutritional status, exercise tolerance, determination of educational needs, and the patient's ability to carry out activities of daily living.[2]

8.2 Areas to be evaluated and reviewed include:[2]

8.2.1 effect on quality of life

8.2.2 pulmonary function assessment, including arterial blood gas analysis

8.2.3 use of medical resources such as hospitalizations, urgent care/emergency room visits, or physician visits

8.2.4 exercise ability

8.2.5 dependence vs independence in activities of daily living

8.2.6 impairment in occupational performance

8.2.7 psychosocial problems such as anxiety or depression

8.2.8 oxygen saturation at rest, with activity, and possibly during sleep

8.2.9 co-morbidity

8.2.10 smoking history

8.2.11 motivation for rehabilitation, including commitment to spending the time necessary for active program participation

8.2.12 current medications

8.2.13 appropriate blood tests

8.2.14 electrocardiogram

8.2.15 chest radiograph

8.2.16 social support

8.2.17 potential need for assistive devices, eg, walker, wheel chair

8.2.18 adherence to recommended treatment modalities

8.2.19 physician support available to patient

8.2.20 availability of transportation and patient/family desire to use what may be available

8.2.21 financial resources

PR 9.0 ASSESSMENT OF OUTCOME:

9.1 Evidence exists for the effectiveness of PR with respect to exercise tolerance, utilization of health care resources, and quality of life.[1,36,66-69] There is some evidence that PR may improve survival in patients with COPD.[36,70-73] The effectiveness of PR can best be established by comparing the baseline condition of the patient to his or her condition as a consequence of participation in the PR program and should involve both qualitative and quantitative measures. Such measurements should include:

9.1.1 indicators of health related quality of life[67,74-81] including a reduction in dyspnea[5,65,67,77,82,83]

9.1.2 enhanced ability to perform activities of daily living including energy conservation[4,84]

9.1.3 increased exercise tolerance and performance[37,41,67,76,77,79,84-88]

9.1.4 decreased respiratory symptoms, eg, frequency of cough, sputum production, wheezing

9.1.5 increased knowledge about pulmonary disease and its management[89-91]

9.1.6 reduced need for medical services including outpatient treatment and hospital admission[70,87,92,93]

9.1.7 increased ventilator-free time in the ventilator-dependent patient

9.1.8 return to productive employment

9.2 Documentation and data collection can develop information regarding the cost-effectiveness of PR.[70,87,92,93]

9.3 The benefit of long-term follow-up, including maintenance programs, should be evaluated.

9.3.1 educational/recreational support group

9.3.2 independent maintenance exercise
9.3.3 scheduled, individualized, on-going exercise/educational input from PR team

10.0 RESOURCES:

10.1 Personnel

The number of disciplines contributing to a PR program varies with the size and scope of the PR program and the availability of those disciplines within the setting. Members might include a respiratory care practitioner, registered or licensed nurse, physical therapist, pharmacist, occupational therapist, dietitian, social worker, exercise physiologist, chaplain, speech therapist, and mental health professional.[2] All personnel should be trained in basic life support techniques and, if possible, advanced cardiac life support.

10.1.1 Medical director: should be a licensed physician with an interest in and knowledge of PR, pulmonary function, and exercise evaluation.

10.1.2 Program director/coordinator: should be trained in health-related profession and have clinical experience and expertise in the care of patients with chronic lung disease. She or he should understand the philosophy and goals of PR and be knowledgeable in administration, marketing, education, patient training, and obtaining reimbursement.

10.1.3 Team members: each member should be well-trained in his or her specialty, demonstrate the ability to establish rapport with and convey the necessary knowledge and skills to patients, and have a good working knowledge of the skills of fellow team members. Each team member should be qualified in their area of expertise to access the patient's needs, provide appropriate intervention, and monitor patient outcomes.[94] The possession of credentials appropriate to each specialty is recommended, as well as appropriate licensing for each state. Persons responsible for pulmonary function testing, blood gas analysis, exercise testing, and those engaged in any patient educational training concerning needed therapy should demonstrate the knowledge and skills specified in the relevant AARC Clinical Practice Guidelines.[33-35,64,95-99] The information and recommendations provided to patients should be evidence-based and consistent across the program. Each team member must be aware of the content of each discipline's educational content.

10.2 Physical facilities

The physical area for PR can vary greatly depending upon program structure, patient population, needs, and resources. The site should provide an appropriate environment with adequate space, few interruptions or other distractions, sufficient lighting and temperature control, and comfortable seating. It is essential to have adequate parking and handicap access.

10.3 Patient education materials[97]

10.3.1 workbooks and videotapes[90]
10.3.2 lung and skeletal models
10.3.3 anatomical posters

10.4 Equipment

10.4.1 stethoscope
10.4.2 manual sphygmomanometer
10.4.3 pulse oximeter[33]
10.4.4 supplemental oxygen source
10.4.5 access to laboratory for arterial blood gas analysis[95]
10.4.6 stopwatch
10.4.7 calibrated cycle ergometer or motorized treadmill (Measured walking distance may be used if an ergometer or treadmill is not available.)[98]
10.4.8 free-weights or elastic bands
10.4.9 patient's own equipment, eg, metered-dose inhaler and spacer, compressor nebulizer for home use[99]
10.4.10 emergency plan and supplies[95]
10.4.11 EKG monitoring during exercise, if indicated, and defibrillation and crash cart[96]
10.4.12 spirometer
10.4.13 peak flow meter

11.0 MONITORING:

11.1 Patient: the following should be monitored at baseline and at appropriate intervals to assure validity of results and appropriateness of intervention:

11.1.1 patient's response to progressive and general reconditioning exercises in conjunction with breathing techniques

11.1.2 patient's oxygen requirements at rest and with exercise

11.1.3 knowledge and skills acquisition: demonstrations and questionnaires should be used to document evidence of change

11.1.4 patient's subjective comments

11.1.5 progress in achieving goals established at baseline

11.2 Patient clinical monitoring during scheduled, supervised session

11.2.1 patient appearance

11.2.2 vital signs

11.2.3 cardiac telemetry, if needed

11.2.4 perceived exertion and dyspnea (eg, use of Borg Scale)

11.2.5 O_2 saturation via oximeter

11.3 PR services: each program should establish clinical indicators that objectively measure the information and instruction provided to the patient and should document the outcomes. Content, goal orientation, and applicability should be reviewed on a regular basis.

12.0 FREQUENCY:

Training and informational components of PR should be delivered in a systematic manner to assure that all patient care issues are addressed. There should be repetition sufficient to ensure retention of information and skills. Giving the patient too much information at one time may cause confusion. Easy-to-read patient education materials should be used to complement and reinforce verbal instructions.[97] Program schedules vary according to staff, facilities, resources, budget, and patient needs.[100] PR services are commonly provided over a period of 12 hours per week for 6 or more weeks, governed by the patient's individual needs.[101] Patients are encouraged, when possible, to participate in an ongoing maintenance exercise program to sustain the training effect.

13.0 INFECTION CONTROL:

13.1 The staff, supervisors, and physicians associated with the PR program should be conversant with "Guideline for Isolation Precautions in Hospitals"[102] and develop and implement policies and procedures for the program that comply with its recommendations for Standard Precautions and Transmission-Based Precautions.

13.2 The program manager and its medical director should maintain communication and cooperation with the mother institution's infection control service and the personnel health service to help assure consistency and thoroughness in complying with the institution's policies related to immunizations, post-exposure prophylaxis, and job- and community-related illnesses and exposures.[103]

13.3 The importance of immunization for influenza[48] and pneumococcal pneumonia,[47] and avoidance of exposure during periods of high incidence of respiratory infections in the community should be stressed to patients. Staff members should receive the influenza vaccination.[104]

13.4 Patients and staff members with signs and symptoms of respiratory infection should avoid contact with patients.

13.5 Adequate handwashing[105] and proper ventilation with prescribed air exchanges should be assured.[106]

13.6 Equipment shared by patients much be cleaned and maintained appropriately. Specific procedures are provided in the 2001 update of static lung volume measurement (Section 13.4-13.7)[107] Proper cleaning methods for the patient's personal therapeutic equipment should be regularly reinforced.[59,97]

14.0 AGE-SPECIFIC ISSUES:

Instructions should be provided and techniques described in a manner that take into consideration the learning ability and communications skills of the patient being served.

14.1 Infant and Neonatal: This Guideline does not apply.

14.2 Pediatric: This Guideline is appropriate for children with indications who can be motivated and who can follow directions.

14.3 Geriatric: This Guideline is appropriate for members of the geriatric population with indications who are motivated and who can follow directions.

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REFERENCES

1. Pulmonary rehabilitation-1999. Official statement of the American Thoracic Society. Am J Respir Crit Care Med 1999; 159(5 Pt 1):1666-1682.
2. American Association of Cardiovascular and Pulmonary Rehabilitation. Guidelines for pulmonary rehabilitation programs. 2nd ed. Champaign, IL: Human Kinetics; 1998.
3. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1995;152(5 Pt 2):S77-S121.
4. Celli BR. Pulmonary rehabilitation in patients with COPD. Am J Respir Crit Care Med 1995;152(3):861-864.
5. Ries AL, Kaplan RM, Limberg TM, Prewitt LM. Effects of pulmonary rehabilitation on physiologic and psychosocial outcomes in patients with chronic obstructive pulmonary disease. Ann Intern Med 1995; 122(1) : 823-832.
6. Tiep BL. Disease management of COPD with pulmonary rehabilitation. Chest 1997;112(6):1630-1656.
7. Cambach W, Wagenaar RC, Koelman TW, van Kiempema AR, Kemper HC. The long-term effects of pulmonary rehabilitation in patients with asthma and chronic obstructive pulmonary disease: a research synthesis. Arch Phys Med Rehabil 1999;80(1):103-111.
8. Foster S, Thomas HM 3rd. Pulmonary rehabilitation in lung disease other than chronic obstructive pulmonary disease. Am Rev Respir Dis 1990;141(3):601-604.
9. Buschbacher R. Outcomes and problems in pediatric pulmonary rehabilitation. Am J Phys Med Rehabil 1995;74(4):287-293.
10. Orenstein DM, Franklin BA, Doershuk CF, Hellerstein HK, Germann KJ, Horowitz JG, Stern RC. Exercise conditioning and cardiopulmonary fitness in cystic fibrosis: the effects of a three-month supervised running program. Chest 1981;80(4):392-398.
11. DeJong W, Grevink RG, Roorda RJ, Kapstein AP, van der Schans CR. Effect of a home exercise training program in patients with cystic fibrosis. Chest 1994;105 (2):463-468.
12. Bach JR. Pulmonary rehabilitation in neuromuscular disorders. Neurology 1993;14:515-529.
13. Stice KA, Cunningham CA. Pulmonary rehabilitation with respiratory complications of postpolio syndrome. Rehabil Nurs 1995;20(1):37-42.
14. Bach JR, Intintola P, Alba AS, Holland IE. The ventilator-assisted individual: cost analysis of institutionalization vs rehabilitation and in-home management. Chest 1992;101(1):26-30.
15. Muir JF. Pulmonary rehabilitation in chronic respiratory insufficiency. 5. Home mechanical ventilation. Thorax 1993;48(12):1264-1273.
16. Craven JL, Bright J, Dear CL. Psychiatric, psychosocial, and rehabilitative aspects of lung transplantation. Clin Chest Med 1990;11(2):247-257.
17. Cooper JD, Trulock EP, Triantafillou A, Patterson GA, Pohl MS, Delaney PA, et al. Bilateral pneumonectomy (volume reduction) for chronic obstructive pulmonary disease. J Thorac Cardiovasc Surg 1995;109(1):106-116; discussion 116-119.
18. Colt HG, Ries AL, Brewer N, Moser K. Analysis of chronic obstructive pulmonary disease referrals for lung volume reduction surgery. J Cardiopulm Rehabil 1997;17(4):248-252.
19. Bernhard J, Ganz PA. Psychosocial issues in lung cancer patients (Part I). Chest 1991;99(1):216-223.
20. Ries AL. Rehabilitation for the patient with advanced lung disease: designing an appropriate program, establishing realistic goals, meeting the goals. Semin Respir Crit Care Med 1996;17:451-463.
21. Emery CF, Leatherman NE, Burker ES, MacIntyre NR. Psychological outcomes of a pulmonary rehabilitation program. Chest 1991;100(3):613-617.
22. Kersten L. Changes in self-concept during pulmonary rehabilitation, Parts 1 and 2. Heart Lung 1990;19(5 Pt 1):456-462 and 1990;19(5 Pt 1):463-470.
23. Folden SL. Definitions of health and health goals of participants in a community-based pulmonary rehabilitation program. Public Health Nurs 1993;10(1):31-35.
24. Hogg JC, Macklem PT, Thurlbeck WM. Site and nature of airways obstruction in chronic obstructive lung disease. N Engl J Med 1968;278(25):1355-1360.
25. Mitchell RS, Stanford RE, Johnson JM, Silvers GW, Dart G, George MS. The morphologic features of the bronchi, bronchioles, and alveoli in chronic airway obstruction: a clinopathologic study. Am Rev Respir Dis 1976;114(1):137-145.
26. Thurlbeck WM. Pathophysiology of chronic obstructive pulmonary disease. Clin Chest Med 1990;11(3):389-403.
27. Enright PL, Hodgkin JE. Pulmonary function tests. In: Burton GG, Hodgkin JE, Ward JJ, editors. Respiratory care: a guide to clinical practice, 4th ed. Philadelphia: JB Lippincott; 1997:225-248.
28. Ries AL, Farrow JT, Clausen JL. Accuracy of two ear oximeters at rest and during exercise in pulmonary patients. Am Rev Respir Dis 1985;132(3):685-689.
29. Steele B. Timed walking tests of exercise capacity in chronic cardiopulmonary illness. J Cardiopulm Rehabil 1996;16(1):25-33.
30. Ries AL. The role of exercise testing in pulmonary diagnosis. Clin Chest Med 1987;8(1):81-89.
31. Jones NL. Clinical exercise testing, 4th ed. Philadelphia: WB Saunders; 1997.
32. Wasserman K, Hansen J, Sue D, et al. Principles of exercise testing and interpretation, 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 1999.

33. American Association for Respiratory Care. AARC Clinical Practice Guideline: Pulse oximetry. Respir Care 1991; 36(12):1406-1409.
34. American Association for Respiratory Care. AARC Clinical Practice Guideline: Postural drainage therapy. Respir care 1991; 36(12):1418-1426.
35. American Association for Respiratory Care. AARC Clinical Practice Guideline: Directed cough. Respir Care 1993;38(5):495-499.
36. American College of Chest Physicians/American Association of Cardiovascular and Pulmonary Rehabilitation Guidelines Panel. Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines. Chest 1997;112(5):1363-1396.
37. Celli BR. Physical reconditioning of patients with respiratory diseases: legs, arms, and breathing retraining. Respir Care 1994;39(5):481-495; discussion 496-500.
38. Ries AL, Ellis B, Hawkins RW. Upper extremity exercise training in chronic obstructive pulmonary disease. Chest 1988;93(4):688-692.
39. Martinez FJ, Vogel PD, DuPont DN, Stanopoulos I, Gray A, Beamis JF. Supported arm exercise vs unsupported arm exercise in the rehabilitation of patients with severe chronic airflow obstruction. Chest 1993;103(5): 1397-1402.
40. Couser JI Jr, Martinez FJ, Celli BR. Pulmonary rehabilitation that includes arm exercise reduces metabolic and ventilatory requirements for simple arm elevation. Chest 1993;103(1):37-41.
41. Lake FR, Hendersen K, Briffa T, Openshaw J, Musk AW. Upper-limb and lower-limb exercise training in patients with chronic airflow obstruction. Chest 1990; 97(5):1077-1082.
42. Dugan D, Walker R, Monroe DA. The effects of a 9-week program of aerobic and upper body exercise on the maximal voluntary ventilation of chronic obstructive pulmonary disease patients. J Cardiopulm Rehabil 1995;15(2):130-133.
43. Dunn AL, Marcus BH, Kampert JB, Garcia ME, Kohl HW 3rd, Blair SN. Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness: a randomized trial. JAMA 1999; 281(4):327-334.
44. Rashbaum I, Whyte N. Occupational therapy in pulmonary rehabilitation: energy conservation and work simplification techniques. Phys Med Rehabil Clin N Am 1996;7:325.
45. Make B. Collaborative self-management strategies for patients with respiratory disease. Respir Care 1994; 39(5):566-579; discussion 579-583.
46. Sturm AW, Mostert R, Rouing PJ, van Klingerin B, van Alphen L. Outbreak of multiresistant non-encapsulated *Haemophilus influenzae* infections in a pulmonary rehabilitation centre. Lancet 1990;335(8683):214-216.
47. Butler JC, Breiman RF, Campbell JF, Lipman HB, Broome CV, Facklam RR. Pneumococcal polysaccharide vaccine efficacy: an evaluation of current recommendations. JAMA 1993;270(15):1826-1831.
48. Rothbarth PH, Kempen BM, Sprenger MJ. Sense and nonsense of influenza vaccination in asthma and chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1995;151(5):1682-1685; discussion 1685-1686.
49. Johnson B. Older adults' suggestions for health care providers regarding discussions of sex. Geriatr Nurs 1997;18(2):65-66.
50. Selecky PA. Sexuality in the pulmonary patient. In: Hodgkin JE, Celli BR, Connors GL, editors. Pulmonary rehabilitation: guidelines to success, 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2000:317-334.
51. Schols AMWJ, Soeters PB, Dingemans AMC, Mostert R, Frantzen PJ, Wouters EF. Prevalence and characteristics of nutritional depletion in patients with stable COPD eligible for pulmonary rehabilitation. Am Rev Respir Dis 1993;147(5):1151-1156.
52. Gray-Donald K, Gibbons L, Shapiro SH, Macklem PT, Martin JG. Nutritional status and mortality in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1996;153(3):961-966.
53. Schols AMWJ, Slangen J, Volovics L, Wouters EF. Weight loss is a reversible factor in the prognosis of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;157(6 Pt 1):1791-1797.
54. Wilson DO, Rogers RM, Sanders MH, Pennock BE, Reilly JJ. Nutritional intervention in malnourished patients with emphysema. Am Rev Respir Dis 1986;134(4):672-677.
55. Hurt RD, Sachs DPL, Glover ED, Offord KP, Johnston JA, Dale LC, et al. A comparison of sustained-release bupropion and placebo for smoking cessation. N Engl J Med 1997;337(17):1195-1202.
56. Silagy C, Mant DC, Fowler G, Lodge M. Meta-analysis on efficacy of nicotine replacement therapies in smoking cessation. Lancet 1994;343(8890):139-142.
57. Fiore MC, for the Guideline Panel and Staff. US Public Health Service Clinical Practice Guideline: Treating tobacco use and dependence. Summary. Rockville, MD: US Dept of Health and Human Services. June 2000. Also published in Respir Care 2000;45(10):1200-1262.
58. Emery CF. Adherence in cardiac and pulmonary rehabilitation. J Cardiopulm Rehabil 1995;15(6):420-423.
59. American Association for Respiratory Care. AARC Clinical Practice Guideline: Discharge planning for the respiratory care patient. Respir Care 1995;40(12):1308-1312.
60. Heffner JE, Fahy B, Hilling L, Barbieri C. Outcomes of advance directive education of pulmonary rehabilitation patients. Am J Respir Crit Care Med 1997;155(3):1055-1059.
61. Heffner JE, Fahy B, Barbieri C. Advance direction education during pulmonary rehabilitation. Chest 1996; 109(2):373-379.
62. Stoller JK. Travel for the technology-dependent individual. Respir Care 1994;39(4):347-360; discussion 360-362.
63. Burns MR. Social and recreational support of the pulmonary patient. In: Hodgkin JE, Celli BR, Connors GL, editors. Pulmonary rehabilitation: guidelines to success, 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2000:465-477.

64. American Association for Respiratory Care. AARC Clinical Practice Guideline: Oxygen therapy in the home or extended care facility. Respir Care 1992;37(8):918-922.
65. Strijbos JH, Postma DS, van Altena R, Gimeno F, Koeter GH. A comparison between an outpatient hospital-based pulmonary rehabilitation program and a home-care pulmonary rehabilitation program in patients with COPD: a follow-up of 18 months. Chest 1996;109(2):366-372.
66. Hodgkin JE. Benefits of pulmonary rehabilitation. In: Fishman AP, editor. Pulmonary rehabilitation. New York: Marcel Dekker; 1996:33-54.
67. Lacasse Y, Wong E, Guyatt GH, King D, Cook DJ, Goldstein RS. Meta-analysis of respiratory rehabilitation in chronic obstructive pulmonary disease. Lancet 1996;348(9035):1115-1119.
68. Donner CF, Muir JF. Selection criteria and programmes for pulmonary rehabilitation in COPD patients. Rehabilitation and Chronic Care Scientific Group of the European Respiratory Society. Eur Respir J 1997;10(3): 744-757.
69. Pulmonary rehabilitation. Thorax 2001;56(11):827-834.
70. Sneider R, O'Malley JA, Kahn M. Trends in pulmonary rehabilitation at Eisenhower Medical Center: an 11-years experience (1976-1987). J Cardiopulm Rehabil 1988;8:453-461.
71. Sahn SA, Nett LM, Petty TL. Ten year follow-up of a comprehensive rehabilitation program for severe COPD. Chest 1980;77(2 Suppl):311-314.
72. Anthonisen NR, Wright EC, Hodgkin JE. Prognosis in chronic obstructive pulmonary disease. Am Rev Respir Dis 1986;133(1):14-20.
73. Burns MR, Sherman B, Madison R, et al. Pulmonary rehabilitation outcome. RT: J Respir Care Pract 1989;2:25-30.
74. Petty T. Pulmonary rehabilitation. Am Rev Respir Dis 1980;122(5 Pt 2):159-161.
75. Guyatt GH, Berman LB, Townsend M, Pugsley SO, Chambers LW. A measure of quality of life for clinical trials in chronic lung disease. Thorax 1987;42(10):773-778.
76. Vale F, Reardon JZ, ZuWallack RL. The long-term benefits of outpatient pulmonary rehabilitation on exercise endurance and quality of life. Chest 1993;103(1):42-45.
77. Goldstein RS, Gort EH, Stubbing D, Avendano MA, Guyatt GH. Randomized controlled trial of respiratory rehabilitation. Lancet 1994;344(8934):1394-1397.
78. Wijkstra PJ, Van Altena R, Kraan J, Otten V, Postma DS, Koeter GH. Quality of life in patients with chronic obstructive pulmonary disease improves after rehabilitation at home. Eur Respir J 1994;7(2):269-273.
79. Troosters T, Gosselink R, Decramer M. Short- and long-term effects of outpatient rehabilitation in patients with chronic obstructive pulmonary disease: a randomized trial. Am J Med 2000;109(3):207-212.
80. Wijkstra PJ, TenVergert EM, Van Altena R, Otten V, Postma DS, Kraan J, Koeter GH. Reliability and validity of the chronic respiratory disease questionnaire (CRQ). Thorax 1994;49(5):465-467.
81. Jones PW, Quirk FH, Baveystock CM, Littlejohns P. A self-complete measure of health status for chronic airflow limitation: the St George's respiratory questionnaire. Am Rev Respir Dis 1992;145(6):1321-1327.
82. Lareau SC, Carrieri-Kohlman V, Janson-Bjerklie S, Roos PJ. Development and testing of the Pulmonary Functional Status and Dyspnea Questionnaire (PFSDQ). Heart Lung 1994;23(3):242-250.
83. Reardon J, Awad E, Normandin E, Vale F, Clark B, ZuWallack RL. The effect of comprehensive outpatient pulmonary rehabilitation on dyspnea. Chest 1994; 105(4):1046-1052.
84. Bendstrip KE, Ingemann Jensen J, Holm S, Bengtsson B. Out-patient rehabilitation improves activities of daily living, quality of life and exercise tolerance in chronic obstructive pulmonary disease. Eur Respir J 1997;10(12):2801-2806.
85. White RJ, Rudkin ST, Ashley J, Stevens VA, Burrows S, Pounsford JC, et al. Outpatient pulmonary rehabilitation in severe chronic obstructive pulmonary disease. J R Coll Physicians Lond 1997;31(5):541-545.
86. Casaburi R, Porszasz J, Burns MR, Carithers ER, Chang RS, Cooper CB. Physiologic benefits of exercise training in rehabilitation of patients with severe chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1997;155(5):1541-1551.
87. Griffiths TL, Burr ML, Campbell IA, Lewis-Jenkins V, Mullins J, Shiels K, et al. Results at 1 year of outpatient multidisciplinary pulmonary rehabilitation: a randomised controlled trial. Lancet 2000;355(9201):362-368.
88. Guell R, Casan P, Belda J, Sangenis M, Morante F, Guyatt GH, Sanchis J. Long-term effects of outpatient rehabilitation of COPD: a randomized trial. Chest 2000;117(4):976-983.
89. Hopp JW, Lee JW, Hills R. Development and validation of a pulmonary rehabilitation knowledge test. J Cardiopulm Rehabil 1989;9:273-278.
90. Morris K, Hodgkin JE, editors. Pulmonary rehabilitation administration and patient education manual. Gaithersburg, MD: Aspen; 1996.
91. Neish CM, Hopp JW. The role of education in pulmonary rehabilitation. J Cardiopulm Rehabil 1988;8:439-441.
92. Lewis D, Bell SK. Pulmonary rehabilitation, psychosocial adjustment, and use of healthcare services. Rehabil Nurs 1995;20(2):102-107.
93. Parker L, Walker J. Effects of a pulmonary rehabilitation program on physiologic measures, quality of life, and resource utilization in a health maintenance organization setting. Respir Care 1998;43(3):177-182.
94. Clinical competency guidelines for pulmonary rehabilitation professionals. American Association of Cardiovascular and Pulmonary Rehabilitation Position Statement. J Cardiopulm Rehabil 1995;15(3):173-178.
95. American Association for Respiratory Care. AARC Clinical Practice Guideline: Sampling for arterial blood gas analysis. Respir Care 1992;37(8):913-917.
96. American Association for Respiratory Care. AARC Clinical Practice Guideline: Resuscitation in acute care hospitals. Respir Care 1993;38(11):1179-1188.

97. American Association for Respiratory Care. AARC Clinical Practice Guideline: Providing patient and caregiver training. Respir Care 1996; 41(7):658-663.
98. American Association for Respiratory Care. AARC Clinical Practice Guideline: Exercise testing for evaluation of hypoxemia and/or desaturation. Respir Care 1992;37(8):907-912.
99. American Association for Respiratory Care. AARC Clinical Practice Guideline: Selection of an aerosol delivery device. Respir care 1992;37(8):891-897.
100. Bickford KS, Hodgkin JE, McInturff SL. National pulmonary rehabilitation survey: update. J Cardiopulm Rehabil 1995;15(6):406-411.
101. Outpatient pulmonary rehabilitation. Local medical review policy. Policy #16.6 Blue Cross of California. (Updated 3/15/00). www.ugsmedicare.com/provider/Lmrp CA/lmrp index.htm#P
102. Garner JS. Guideline for isolation precautions in hospitals. Part I. Evolution of isolation practices. Hospital Infection Control Practices Advisory Committee. Am J Infect Control 1996 Feb;24(1):24-31.
103. Bolyard EA, Tablan OC, Williams WW, Pearson ML, Shapiro CN, Deitchmann SD. Guideline for infection control in healthcare personnel, 1998. Hospital Infection Control Practices Advisory Committee. Infect Control Hosp Epidemiol 1998;19(6):407-463. [Erratum in: Infect Control Hosp Epidemiol 1998;19(7):493.]
104. US Center for Disease Control and Prevention. Prevention of influenza: recommendations of the Advisory Committee on Immunization Practices. MMWR 2000;49(RR-03):1-38.
105. Larson EL. APIC guideline for handwashing and hand antisepsis in health care settings. AM J Infect Control 1995;23(4):259-269.
106. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities, 1994. Centers for Disease Control and Prevention. MMWR Morb Mortal Wkly Rep 1994 Oct 28;43(RR-13):1-132 or Federal Register 1994;59(208):54242-54303.
107. American Association for Respiratory Care. AARC Clinical Practice Guideline. Static lung volume, 2001 revision and update. Respir Care 2001;46(5):531-579.

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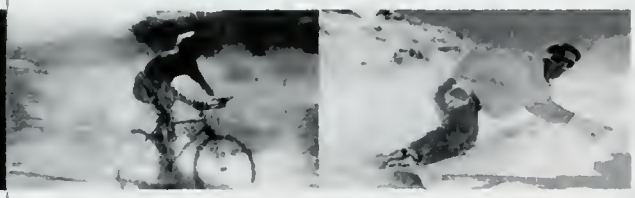

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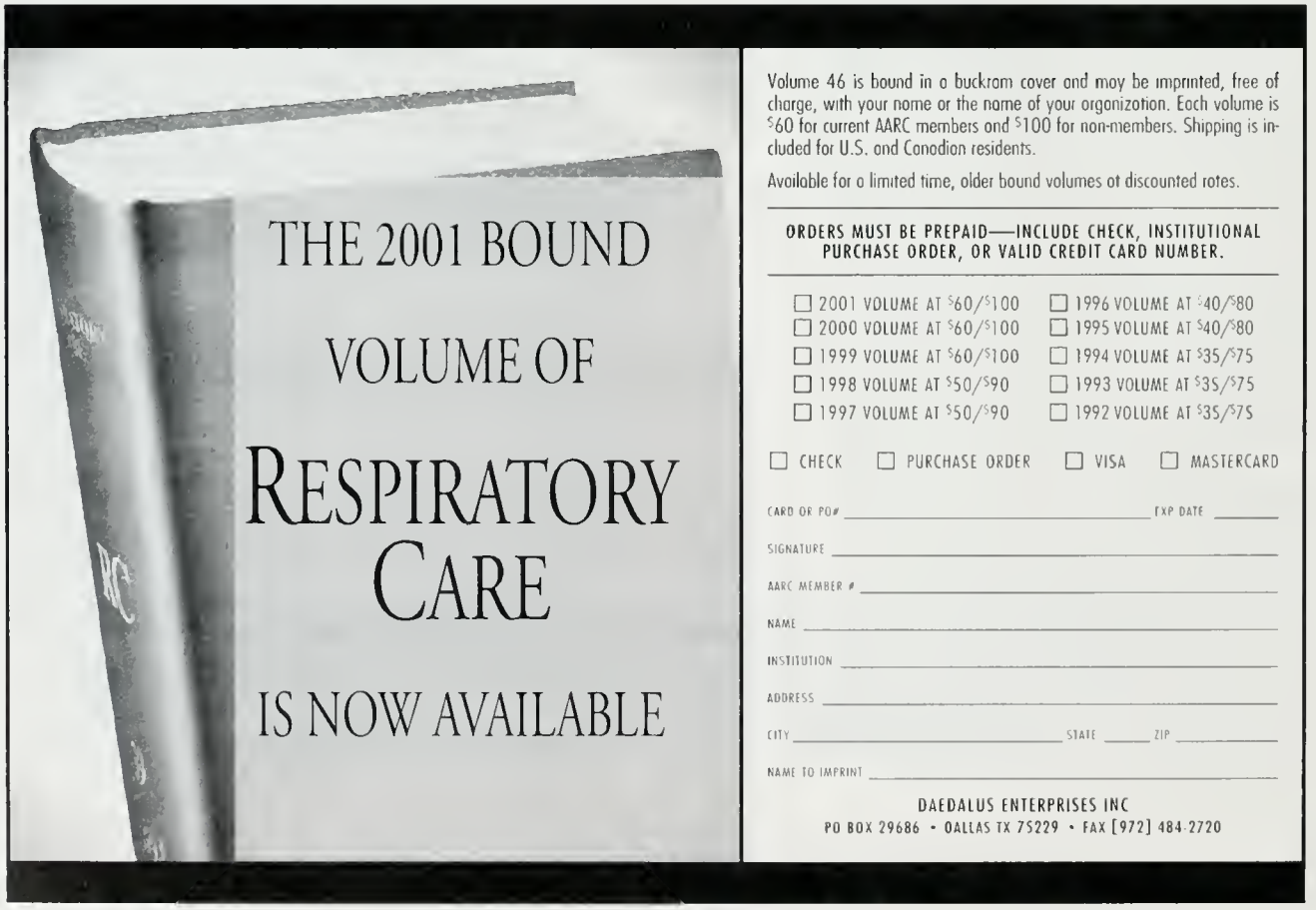

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SATURDAY, Dec. 1

- ❑ 1. Keynote Address, D. Satcher
- ❑ 5. Tracking Flow of Residents, D.N. Muse
- ❑ 6. (6.) Role of Educators, E.A. Becker &
  (7.) Role of Managers, K.J. Stewart &
  (8.) Panel Discussion, M.Traband
- ❑ 9. Errors, Law and RCPs, A.L. DeWitt
- ❑ 10. Human Error: Changing Our Perceptions, J.W. Salyer
- ❑ 11. Patient Abuse in the Home: What Is It and What Are Your Responsibilities? D. Roberts
- ❑ 12. Assessing the Right Competencies for Home Care Therapists, S.L. Moreau
- ❑ 13. Discharging the Complex Pediatric Patient to the Home, J.P. Stegmaier
- ❑ 14. Does Economics Drive Home Care? R.W. McCoy
- ❑ 15. Surfactant Therapy, R.J. Rodriguez
- ❑ 16. Neonatal Update: Inhaled Nitric Oxide in the Neonate, S.R. Seidner
- ❑ 17. Mgt. Strategies for Prevention of BPD, R.J. Rodriguez
- ❑ 18. Overview of Dying and End-of-Life Care in America, P.A. Selecky
- ❑ 19. Withholding and Withdrawing Ventilation, R. Acevedo
- ❑ 20. Role of the Respiratory Therapist in Palliative Care, H.M. Sorenson
- ❑ 21. (21.) Building Coalitions, J.B. Fink
  (22.) Reaching Patients and Physicians with "Baylor's Rules of Two"!, G.E. Lawrence
- ❑ 23. (23.) Regional Asthma Center, L. Parish-Cooper
  (24.) Promoting Public Awareness: Successful Strategies, S. Blonshine
- ❑ 25. (25.) Sleep Disordered Breathing, M.B. Dunning III
  (26.) Protocols for Standardized Sleep Testing, R.N. Turner
- ❑ 27. (27.) New Polysomnograph Technologist Training Guidelines, R.N. Turner
  (28.) Survival Techniques for Shift Workers, M.B. Dunning
- ❑ 31 A. Issues & Trends in Allied Health Leadership, M. Harrington, Developing Your Career for Maximum Professional Effectiveness, M. W. Runge
  Breakout #1: Leadership in Clinical Practice, G. Gaebler
  Breakout #3: Leadership in Management, J.D. Kimble (Needs overtime tape)
- ❑ 31 B. Developing Professional Leadership, R. Krueger
  Success Stories, J.C. Bolden , D. Johnson
- ❑ 31 C. #2: Leadership in Clinical Research, A.B. Adams &
  #4: Leadership in Education, L. Van Scoder
- ❑ 34. Humor at the Bedside, M. Graves

SUNDAY, Dec. 2

- ❑ 35. Egan Lecture — Chronic Obstructive Pulmonary Disease - On the Exponential Curve of Progress, J.E. Heffner
- ❑ 36. Pulmonary Rehabilitation, B.W. Carlin
- ❑ 37. High Frequency Oscillatory Ventilation for ARDS: State of the Art 2001, S. Derdak
- ❑ 38. "The Ins and Outs" of Lung Transplantation, S.M. Levine MD, C.G. Durbin Jr.
- ❑ 39. Perspectives from Respiratory Care and Nursing, P. Carroll, & A.L. DeWitt Jr.
- ❑ 40. Risk Management and Respiratory Care, S.J. Price
- ❑ 41. Competency: Is Your Staff Ready? J.R. Edens
- ❑ 42. Managing Protocols Based on CPGs, E. Hoisington
- ❑ 45. A New Opportunity, C.M. Kercsmar
- ❑ 46. Questions for the Attorney, A.L. DeWitt
- ❑ 47. JCAHO: Showcasing Your Best, R.M. Ford
- ❑ 48. Justifying Human Resources: Best Use of a Valuable Asset, R.M. Ford
- ❑ 49. (49.) It's All Black and White, B. Yawn &
  (50.) Managing to Breathe via Internet, C. Olson
- ❑ 51. The Dirty Dozen, N. Sander
- ❑ 52. Selecting Clinical and Functional Outcomes in LTOT, T.L. Petty
- ❑ 53. Selecting Fiscal and Satisfaction Outcomes in LTOT, T.W. Buckley
- ❑ 54. Measuring and Collecting Outcome Data, D.C. Shelledy
- ❑ 55. Using LTOT Outcome Data, R. Fary
- ❑ 56. Outcome Data Panel Discussion, T.W. Buckley, R. Fary, T.L. Petty, and D.C. Shelledy
- ❑ 57. Smoking Cessation, D.D. Gardner
- ❑ 58. New Strategies for Treating Tobacco Use and Dependence, R.C. Cohn
- ❑ 59. How Respiratory Care Managers Can Implement Tobacco Education Programs for Community-Wide Effect, S.M. Ciarlariello
- ❑ 60. Secondhand Smoke — What Can Health Care Professionals Do About It? A. French, B. Huffman
- ❑ 61. (61.) Evolution and Rationale of the Low VT Protocol, R. Brower
  (62.) Practical Aspects of Implementing the Low VT Protocol, R. Kallet
- ❑ 63. (63.) Can the ARDS Network Protocol Be Modified?, N.R. MacIntyre &
  (64.) How and Why to Use Recruitment Maneuvers in Low VT Ventilation, N.R. MacIntyre
- ❑ 65. (65.) Normal Mechanisms of Airway Clearance and Problems from the ICU to Home, B.K. Rubin &
  (66.) Positioning vs. Postural Drainage, J. Fink &
  (67.) Breathing Maneuvers, M. MacIllwaine
- ❑ 68. (68.) Positive Pressure Techniques for Airway Clearance, M.J. Mahlmeister &
  (69.) High Frequency Oscillation of the Airway and Chest Wall, J. Fink &
  (70.) Airway Clearance and the Artificial Airways, R. Lewis
- ❑ 71. (71.) Pharmaceutic Approach to Airway Clearance, B.K. Rubin &
  (72.) Strategies for the Pediatric Patient, K.L. Davidson &
  (73.) Airway Clearance Strategies for the Vent Patient at Home, C. Lapin
- ❑ 74. RSV: Management and Treatment Advances, C.M. Kercsmar
- ❑ 75. Test Your Skills on the State of the Profession, M.F. Traband
- ❑ 76. How to Survive as a Multi-Department Manager, S.J. Price
- ❑ 79. Technological Advances in Noninvasive Respiratory Monitoring, G. Devine
- ❑ 80. Monitoring on the General Floor: What's the Future Going To Be Like? P.T. Sharkey
- ❑ 81. A Proposal for Evidence-Based Practice in Neonatal Resuscitation, W.D. Rich
- ❑ 82. Classifying Asthma Patients through Symptom Analysis, T.J. Kallstrom
- ❑ 83. How Not to Run a Respiratory Care Service, K.L. Shrake

MONDAY, Dec. 3

- ❑ 84. Kittredge Lecture — Mechanical Ventilation: How Did We Get Here and Where Are We Going?, R.D. Branson
- ❑ 85. Intrapulmonary Percussive Ventilation, C. Diaz
- ❑ 88. Exercise Training: Are You Training Your Patients at the Right Exercise Target? B.W. Carlin
- ❑ 89. Water Exercise in Pulmonary Rehabilitation Programs, M.W. Millard
- ❑ 90. The Impact of the Institute of Medicine's Report for Respiratory Care Providers, S.P. Giordano
- ❑ 91. Evidence-Based Respiratory Care, D.J. Pierson
- ❑ 92. Managing Chronic Medical Conditions in the New Care Delivery Framework, P.J. Dunne
- ❑ 93. (93.) Applying Case Management Principles in Home Care, J.P. Stegmaier &
  (94.) Developing a Cost Model For Delivery of LTOT, T.W. Buckley
- ❑ 95. (95.) What I Wish All Home Care Therapists Knew, B. Rogers, &
  (96.) LTOT Utilization: The Bigger Picture! G. Spratt
- ❑ 97. (97.) Modifying Treatment Plans For the Aging Lung, T.S. LeGrand &
  (98.) Seeing Health Care through the Eyes of the Elderly, H.M. Sorenson

- ❑ 99. (99.) Caring for the Older Pneumonia Patient, T.S. LeGrand & (100.) Senior Pulmonary Assessment, H.M. Sorenson
- ❑ 101. Interpretation of PFTs by RCPs, J.O. Nilsestuen
- ❑ 102. Results of a National Study, J.M. Boyle
- ❑ 103. Agency Updates ñ Through the eyes of the AARC, ARCF, CoARC and NBRC, C.P. Wiezalis, M.T. Amato, M. Welch Jr, and J. Long-Goding
- ❑ 104. Munchausen by Proxy, D. Roberts
- ❑ 105. Opening the Black Box during Mechanical Ventilation, M. Amato
- ❑ 107. (107.) Documenting Respiratory Practice Beyond the USA, J. Long-Goding H.L. Garza & (108.) Comparison of Two Methods for Administration of Inhaled Salbutamol, H.L. Molina
- ❑ 109. (109.) The Brazilian Experience, L.G. Ghion & (110.) The United Kingdom Experience, A. Hynes
- ❑ 111. (111.) The Italian Experience, S. Thompson & (112.) The Japanese Experience, H. Etchuya
- ❑ 113. (113.)The North America Experience, A. King & (114.) The Personal Experience — A Patient's View, B. Rogers, & Panel Discussion
- ❑ 115. Creating an Effective Multi-Cultural Work Environment, K.L. Shrake
- ❑ 116. Bronchiolitis Care Path, J.W. Salyer
- ❑ 117. Pediatric Asthma: Success with Therapist-Driven Protocols, T.R. Myers
- ❑ 118. Developing a Pediatric Consult Program, J.W. Salyer
- ❑ 119. Lung Protective Strategies for Status Asthmaticus, M.K. Brown
- ❑ 120. Acute Care of the Adult Asthmatic, J.I. Peters
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TUESDAY, Dec. 4

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- ❑ 140. Diagnostic Testing: Pre- and Post-Lung Transplant, C.M. Foss
- ❑ 141. Lung Transplantation from the Patient's Perspective, M. Pierce
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- ❑ 147. Using Protocols in Respiratory Home Care, G. Spratt
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- ❑ 155. Pulmonary Hypertension and the Respiratory Therapist, R.L. Rosenblatt
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- ❑ 169. Neonatal Transport Cases, S. Segovia
- ❑ 170 A&B (2 Tapes) Allergic Disorders: The Child with Rhinitis and Asthma, B. Martin
- ❑ 171. What's New in CPR ALS? T.A. Barnes
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SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocal valuation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

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We encourage electronic submission. Send all submissions to barcus@aarc.org. They will be acknowledged immediately via return e-mail. Mailed confirmation copies are not necessary, except that we ask you to fax a hard copy of your abstract so we can check it for accuracy against the electronic version. The fax number is 972-484-6010 (marked to the attention of Linda Barcus). **Paper size** should be 8.5" x 11" (approx 21 x 28 cm), with all 4 margins set to 1.5" (approx 4 cm). **Figures & Tables.** Save the text portion of your abstract in a Microsoft Word compatible file format. Excel is preferred for any accompanying graphics. If you do not have an Excel compatible format, save your graphics as TIFF or EPS files. Simply renaming a file with a .tif or .eps suffix will not work. Do not send Lotus files. Please send the figure as an attachment separate from the text. We can reduce it to the correct size, but unless you send the figure by itself, all we will have is an image that cannot be reproduced. The fax copy you send will show us the exact placement and size of the figure. Remember that all figures and tables will be greatly reduced in size, so keep them simple enough to be readable.

Deadline. The mandatory Deadline is April 30, 2002 (postmark). Authors will be notified of acceptance or rejection *by letter only*. These letters will be mailed by June 15, 2002.

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Electronic Oxygen Conserver. CHAD Therapeutics Inc announces the introduction of its new OXYMATIC 411 electronic oxygen conserver. This is the first conserver from CHAD to deliver a pulse of oxygen on every breath at all delivery settings. It is an all-in-one unit has a built-in brass regulator and offers all the features found in their OXYMATIC 401 conserver, including: uses standard cannula; 1-6 L/min flow equivalency settings; continuous flow setting at 2 L/min; simplified patient operation. For more information from CHAD Therapeutics, circle number 171 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Spirometer. SDI Diagnostics introduces its new Spirolab II Spirometer, saying that it offers maximum portability in an accurate, easy-to-operate instrument. The large, bright LCD display shows spirometry testing real-time and includes pre- and post-drug comparisons with flow/volume loop overlays, according to the manufacturer. Spirolab II measures over 30 different parameters with automatic interpretation and test quality control prompts. Printout capabilities are from the silent internal thermal printer or via a direct link to an inkjet printer. SDI claims that its full user-friendly keyboard with dedicated function keys plus alphanumeric data entry allows patient retrieval by number or name. The Spirolab II uses easily available mouthpieces or filters and, considering its wealth of features, is affordably priced, according to the manufacturer. For more information from SDI Diagnostics, circle number 172 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Forehead Sensor. Nellcor has introduced the MAX-FAST Forehead Sensor as part of the new OxiMax Pulse Oximetry System. According to the manufacturer, this new forehead sensor displays changes in S_{pO2} faster than digit sensors during poor perfusion conditions. The MAX-FAST adhesive forehead sensor works with any OxiMax monitor. The digital memory chip embedded in the sensor enables it to "talk" with the OxiMax monitor, where clinical information is displayed. The MAX-FAST forehead sensor is the first sensor approved by the FDA for use on mechanically ventilated patients, according to Nellcor. For more information from Nellcor, circle number 173 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

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New Deadline for Submission of OPEN FORUM Abstracts Is

May 17, 2002

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Scheduled Professor's Rounds 2002

Withholding and Withdrawing Life Support in the ICU — Gordon D Rubenfeld MDMSc / Richard D Branson BA RRT FAARC — Videotape Available

Weaning from Mechanical Ventilation: New Insights, New Guidelines — Neil R MacIntyre MD FAARC/ Dean R Hess PhD RRT FAARC — Live April 16; Audio May 14

Neonatal and Pediatric Ventilators: What's the Difference? — Mark J Heulitt MD FAARC/ Richard D Branson BA RRT FAARC — Live May 21; Audio June 25

Ventilator Graphs: What's With That Wave? — Jon O Nilsestuen PhD RRT FAARC/ Richard D Branson BA RRT FAARC —Live July 16; Audio August 13

Talking with Patients and Families About Death and Dying— Helen M Sorensen MA RRT FAARC/ David J Pierson MD FAARC —Live August 20; Audio September 17

Pressure vs Volume Ventilation: Does It Matter? — Robert S Campbell RRT FAARC/ Richard D Branson BA RRT FAARC — Live September 10/ Audio October 8

Inpatient Management of COPD — Randall Rosenblatt MD/ David J Pierson MD FAARC — Live October 22; Audio November 12

High-Frequency Oscillatory Ventilation — Thomas E Stewart MD/ Richard D Branson BA RRT FAARC — Live November 19; Audio December 10

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
- — Current job listings
- — American Respiratory Care Foundation fellowships, grants, & awards
- — Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
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- — Subject and Author Indexes
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Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

Committee on Accreditation for Respiratory Care
http://www.coarc.com

The National Board for Respiratory Care— Examination Fees for 2002

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Calendar of Events

Date	AARC & State Society Programs	Contact
May 13–15	MSRC's Annual Spring Fling, **Lewiston, ME**	Diane Haskell (207) 596-8872 or Dhaskell@nehealth.org; Bobbie Crockett (207) 262-2214 or roberta.crockett@sjhhealth.com; www.mesrc.org
June 11–13	New Jersey and New York State Societies for Respiratory Care, 15th Annual Spring Forum; **Round Top, NY**	Ken Wyka (NJ) at (201) 725-2528; or Bob Fluck (NY) at (315) 464-5580
June 14–16	AARC Summer Forum; **Vail, CO**	AARC, (972) 243-2272, www.aarc.org
June 16	AARC/EPA Asthma and Indoor Air Environment Workshop; **Keystone, CO**	AARC, (972) 243-2272, www.aarc.org
June 16–17	AARC Asthma Disease Management Course; **Vail, CO**	AARC, (972) 243-2272, www.aarc.org
June 19–21	ISRC 34th Annual Convention & Exhibition; **Oakbrook Terrace, IL**	General information, Kelli DeBerry (847) 981-3581; vendors, Larry Dastych (847) 618-3180
June 27	Asthma Information Review course; **Grand Rapids, MI**	Michigan Society for Respiratory Care, (734) 677-6772, www.michiganrc.com
June 27–29	34th Annual California Society for Respiratory Care Convention; **Universal City, CA**	CSRC Executive Office (888) 730-CSRC or www.csrc.org
June 27–29	34th Annual CSRC State Convention; **Universal City, CA**	CSRC (818) 247-2053, www.csrc.org
July 17–19	TSRC 31st Annual Convention and Exhibition; **San Antonio, TX**	TSRC Executive Office (972) 680-2455
July 27	Asthma Information Review course; **Flint, MI**	Michigan Society for Respiratory Care, (734) 677-6772, www.michiganrc.com
Aug. 1–3	OSRC's Contemporary Concepts in Respiratory Care; **Independence, OH**	Tom Kallstrom at tom.kallstrom@fairviewhospital.org; or Jeffrey Davis at davisj@ccf.org
Aug. 1–4	Georgia Society for Respiratory Care's Summer Meeting; **St. Simons, GA**	David Ellwanger, (770) 991-8558, ellwanger_david@promina.org
Aug. 14–16	31st TriState Respiratory Care Conference; **Biloxi, MS**	Linda Berry (205) 343-8475; www.tsrcc.org
Sept. 5–6	Vermont/New Hampshire Society for Respiratory Care Annual Meeting; **Killington, VT**	Xan Gallup (800) 333-8095, ext. 360 or xgallup@merriam-graves.com
Sept. 5–7	TSRC North Region – Pineywoods District 16th Annual Fall Seminar; **Lufkin, TX**	Pam Hollis (409) 639-7006
Sept. 11–12	MSRC 25th Annual State Conference; **Sturbridge, MA**	Frances Smith (508) 833-9893, fsmith91@hotmail.com
Sept. 12–13	PSRC Southwest District's 29th Annual Seminar in Pulmonary and Sleep Medicine; **Pittsburgh, PA**	Gail Varcelotti (724) 941-8792, SWDistrict@psrc.net
Sept. 12–13	Alabama Society for Respiratory Care Annual Meeting; **Birmingham, AL**	Bill Pruitt (251) 434-3405 or wpruitt@jaguar1.usouthal.edu
Sept. 19–20	Kansas Respiratory Care Society Western Kansas Conference; **Hays, KS**	www.krcs.org or e—mail Julia Downs at jddrrt@yahoo.com
Oct. 5–8	AARC 48th International Respiratory Congress; **Tampa, FL**	AARC, (972) 243-2272, www.aarc.org

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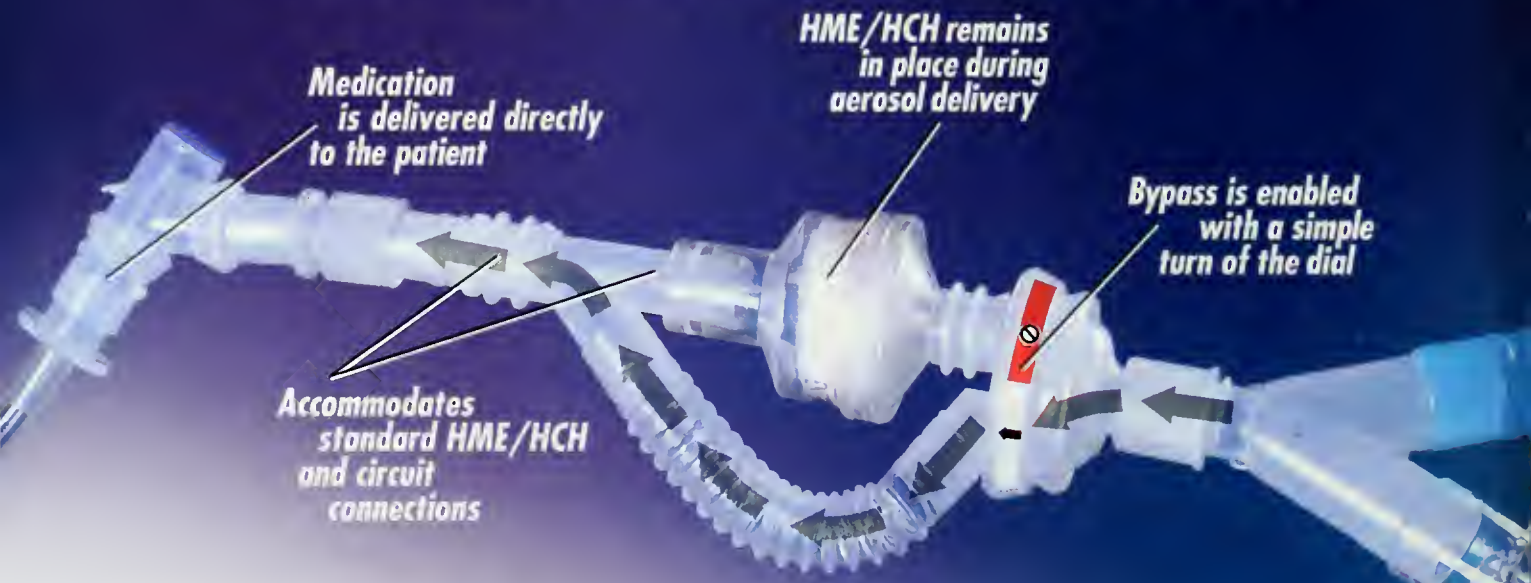
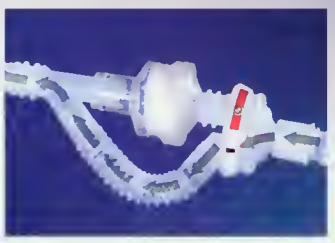
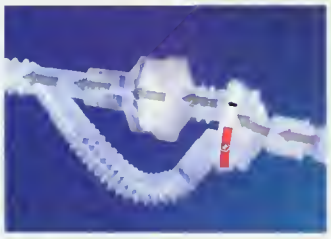
The aerosol delivery breakthrough with no breaks in the circuit


Medication is delivered directly to the patient
HME/HCH remains in place during aerosol delivery
Bypass is enabled with a simple turn of the dial
Accommodates standard HME/HCH and circuit connections



Bypass Position



HME/HCH Position

Until now, providing aerosolized medications to mechanically ventilated patients meant removing the HME/HCH – a procedure that can increase the risk of alveolar derecruitment, drying of airway secretions, and contamination to both patients and clinicians.

Now you can reduce these risks with new CircuVent™ — the breakthrough device that lets you provide aerosol *without* breaking the circuit!

You'll improve patient safety and comfort, save time, and reduce costs associated with repeated HME/HCH removal and replacement.

For more information, call DHD Healthcare toll-free today: **1-800-847-8000.**

DHD Healthcare

Innovations for respiratory care

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Contraindicated in patients who cannot tolerate additional dead space
Non-conductive • Non-sterile • Single patient use • Do not autoclave
CAUTION: Federal (USA) law restricts this device to sale by or on the order of a physician

CircuVent is a trademark of DHD Healthcare Corporation ©2001 DHD Healthcare Corporation

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Circle 108 on product information card